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(British Medical Association)

Second Session: Dunedin, February 3 to 10, 1927

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SECTION IV.—PATHOLOGY AND BACTERIOLOGY.

TUBERCULOUS LESIONS MET WITH IN EIGHT HUNDRED AUTOPSIES WITH DEDUCTIONS THEREFROM.

By J. BURTON CLELAND, M.D.,
Professor of Pathology, University of Adelaide; Honorary Pathologist, Adelaide Hospital.

(*Continued from page* 448.)

TABULATED RESULTS.

No Tuberculous Lesions Detected.

There were five hundred and twenty-seven subjects or 66% in whom no tuberculous lesions were detected.

Healed Tuberculosis.

In one hundred and three subjects or 13% the tuberculosis was healed.

In a considerable number of *post mortem* examinations the only evidence obtainable of a previous infection with the tubercle bacillus is in the shape of small areas of calcification with usually some fibrosis around them situated in various lymphatic glands and in the lungs. Here the small area of caseation resulting from the tuberculous process has finally had calcium salts deposited in it. This is a process that must take time. I have seen such calcification, however, in patients aged only twenty, who had presumably been affected in infancy. Calcified areas found in old people, seventy or eighty years of age, probably represent in many cases at least infections contracted in childhood. Any tubercle bacilli originally present in the area now calcified will presumably be dead. To test this a guinea pig was inoculated from the calcified mesenteric gland of a man, aged fifty-one, born in Australia, and the animal showed no evidence of tuberculosis when killed. Another guinea pig was inoculated with material from a small calcified area in a lung (not at the apex) of a man of sixty-five, born in Australia, who had syphilitic aortitis, the result being negative.

Often round these calcareous areas there is only a thin capsule of fibrous tissue as for instance in many calcified mesenteric glands. The tuberculous process seems in fact to have entirely died out. Such cases I have classified under the heading of "Healed Tuberculosis," believing that in most cases living tubercle bacilli are no longer present. It is possible, however, that in some of these cases some living bacilli may still be present.

Calcified Areas, Sometimes only the Size of a Grain of Wheat, in Lymphatic Glands.

Calcified or calcified and fibrosed glands at the root of the lung	24
Calcified mesenteric glands	15
Calcified mediastinal or tracheal glands	5
Calcified mesenteric glands and gland at the root of the lung	2
Calcified mediastinal glands and glands at the root of the lung	1
Calcified glands at the root of the lung and in the portal fissure	1
Total	48

Calcified Areas, Sometimes Mere Specks the Size of Half a Grain of Wheat, with more or less Surrounding Slight Fibrosis in the Lung Tissue.

Small calcified area or speck at one apex with or without fibrosis	20
Similar lesions affecting both apices	2
Small calcified areas or specks elsewhere in the lung with or without fibrosis	13
Old tuberculous scars at both apices	1
Small calcified area at the apex and another elsewhere	2
Scars at both apices and a calcified nodule elsewhere in the lung	1
Slight scarring at an apex, adhesions and pleuritic thickening	1
Apical scars or calcified speck and calcified gland at the root of the lung	4
Calcified speck in a middle lobe with calcification in a gland at the root of a lung	1
Thickened pleura at the apex with a calcified gland at the root of the same lung	1
Slight pleuritic thickening at the apex, calcification of a gland at the root of the lung on the same side and calcification of the mediastinal glands	1
Old fibrosis of the apex and calcification in a mediastinal gland	1
Scars at both apices, one calcified, with calcified mesenteric gland	1
Slight puckering at an apex (? tuberculous) with calcified mesenteric gland	1
Calcified area in the lung with calcified mesenteric glands	2
Calcified nodule in the lower part of one upper lobe with a calcified gland at the root of the lung and a calcified millet-seed-sized area in the liver (? tuberculous)	1
Total	53

Healed Tuberculosis Elsewhere.

Old healed tuberculosis of the bodies of the dorsal vertebræ	1
Healed or healing tuberculosis of lumbar vertebræ with a small collection of pus near it and the healed track of an ileo-psoas abscess *plus* calcified specks in a gland at the root of one lung	1
Total	2

The above cases total one hundred and three. In forty-eight cases the lesions were confined to lymphatic glands; in fifty-three lesions were present in the lungs and in thirteen of these in lymphatic glands as well. Two represent healed or healing tuberculosis of the vertebræ.

Quiescent or Healing Tuberculosis.

There were eleven instances of quiescent or healing tuberculosis.

In the following the condition is considered as being stationary and perhaps as remaining so for many years, but the tubercle bacilli are probably still alive and capable of doing further damage if the old focus is injured as by an adjacent patch of pneumonia.

Fibrosed area with streaks of caseation or dense fibrosis only, at one apex 3
Scar at the left apex with a caseo-calcareous bar in the subpleural tissue near the right apex 1
Some fibrosis with small caseous specks at both apices, pleuritic adhesions 1
Smooth slight cavity at the right apex with calcareous material in its wall and no involvement of the lung beyond (? healed) 1
Puckering at the right apex with a number of hard tuberculous nodules *plus* caseous foci in the glands at the root of the lung 1
Healed tuberculous lesions of both pleuræ and lungs *plus* caseation in the glands at the roots of a lung *plus* tuberculous right hip (healed), tuberculosis in a suprarenal gland, and probably also in the left kidney 1
Small grey caseous specks in a gland at the root of a lung and a small caseous speck in the spleen .. 1
Calcified specks in a gland at the root of a lung *plus* healed or healing tuberculosis of lumbar vertebræ with an adjacent small collection of pus and the healed track of a psoas abscess 1
Small caseous foci in a jejunal mesenteric gland 1

Total 11

Extensive Pulmonary Tuberculosis.

These cases numbered fifty-one or 6·4%. They consist of those in which pulmonary tuberculosis was present with in most cases cavity formation and invasion of both lungs. With one or two exceptions the patient's death has been due to the tuberculosis.

Extensive pulmonary tuberculosis usually with cavity formation 18
Extensive pulmonary tuberculosis *plus* tuberculous ulcers of the intestine (in two of the patients the tuberculous ulcers had perforated) 7
Extensive pulmonary tuberculosis *plus* tuberculous ulcers of the intestine and tuberculous laryngitis or tuberculosis of the epiglottis 4
Extensive pulmonary tuberculosis *plus* calcified mesenteric glands 3
Extensive pulmonary tuberculosis *plus* tuberculous ulcers of the intestines *plus* calcified tuberculosis of a mediastinal gland or gland at the root of a lung 3
Extensive pulmonary tuberculosis *plus* tuberculous ulcers of the intestines *plus* calcified and caseous mesenteric glands *plus* secondary tuberculous peritonitis 1
Extensive pulmonary tuberculosis *plus* calcified specks in a gland at the root of the opposite lung 1
Extensive pulmonary tuberculosis *plus* tuberculous meningitis 1
Extensive pulmonary tuberculosis *plus* miliary tuberculosis of the lungs and organs 1
Caseous area in the right lung *plus* tuberculous peritonitis *plus* miliary tuberculosis of the liver, spleen and kidneys 1
Extensive pulmonary tuberculosis *plus* caseous bronchial glands *plus* miliary tuberculosis of the spleen, liver and peritoneum 1
Extensive one-sided tuberculous pleurisy with early tuberculosis of the lung 1
Extensive silicosis with cavitation and caseation at the right apex (death due to silicosis) 1
Silicosis with cavitation, but no obvious caseation or tubercles, but with tuberculous ulcers of the intestine and a calcified speck in a gland at the root of a lung 1
Extensive pulmonary tuberculosis *plus* calcified specks in glands at the root of a lung *plus* neck sinuses leading to healed tuberculous cervical glands .. 1
Caseated area in the lung with abundant racemose tubercles *plus* a mass of tubercles in a mediastinal gland *plus* caseated glands in the neck *plus* caseated mesenteric glands and tuberculous peritonitis with caseated masses *plus* caecal ulceration
Extensive pulmonary tuberculosis *plus* tuberculous ulcers of the intestine *plus* caseated mesenteric glands *plus* caseated glands at the root of the lung *plus* miliary tuberculosis of the spleen 1
Extensive pulmonary tuberculosis *plus* tuberculous ulcers of the intestine *plus* calcified mesenteric gland *plus* tuberculosis of the epididymis and prostate 1
Extensive pulmonary tuberculosis *plus* tuberculous ulcers of the intestine *plus* tuberculous mesenteric glands *plus* amyloid disease 1
Caseous tuberculosis of the lungs (bases almost solid) *plus* caseous deposits in the pleura, liver and spleen 1
Tuberculosis of the middle lobe of the right lung and some small foci in other parts of this lung *plus* tubercles on the pleura, *plus* tuberculous mediastinal gland *plus* early tuberculous peritonitis .. 1

Total 51

Tuberculous Meningitis.

There were six subjects with tuberculous meningitis.
Tuberculous meningitis *plus* tuberculous glands at the root of the lung (in a girl, 1¼ years) 1
Tuberculous meningitis *plus* caseating focus in a gland at the root of the lung *plus* miliary tuberculosis of the lung, liver and spleen (in a girl, 6¾ years) 1
Tuberculous meningitis *plus* a small caseating nodule with some miliary tubercles round it in the upper part of the left lung (in a boy, 4 years) 1
Tuberculous meningitis *plus* a small focus in the lower part of one upper lobe and a smaller one in the lower lobe *plus* tuberculosis of the right kidney (in a woman, 24 years) 1
Tuberculous meningitis *plus* tuberculous mesenteric glands *plus* tuberculosis of the peritoneum *plus* tuberculous nodules of the *dura mater* (in a girl, 3¼ years) 1
Tuberculous meningitis *plus* a large caseated area in the liver (in a man, 36 years) 1

Total 6

Miliary Tuberculosis.

Four subjects were found to have miliary tuberculosis.
Miliary tuberculosis of the lungs and kidneys *plus* fibro-caseous mediastinal glands (in a girl, 17 years) 1
Miliary tuberculosis of the lungs and kidneys *plus* tuberculous mediastinal and mesenteric glands (in a girl, 11 years) 1
Miliary tuberculosis of the lungs and peritoneum *plus* caseous glands at the root of the lung (in a woman, 22 years) 1
Miliary tuberculosis of the lungs *plus* miliary tuberculous peritonitis *plus* tuberculosis of the mesenteric glands and of the peritoneal aortic glands and mediastinal glands and glands at the roots of the lungs (in a woman, 21 years) 1

Total 4

Extensive, Not Specially Pulmonary Tuberculosis.

The number in the group of extensive tuberculosis not specially pulmonary in distribution, death usually due to the tuberculosis, was seven.

Tuberculosis of the endometrium *plus* tuberculous peritonitis *plus* tuberculous glands at the root of the lungs *plus* miliary tuberculosis of lungs (in a woman, 25 years) 1

Tuberculosis of the right hip *plus* tuberculous peritonitis *plus* tuberculous ulcers of the intestine *plus* caseating glands at the root of the lung (in a girl, 10¾ years) 1

Massive tuberculosis of the last fifteen centimetres of the ileum *plus* caseating mesenteric glands *plus* tuberculous gland at the root of the lung (in a boy, 9¼ years) 1

Caseous mesenteric glands and glands at the roots of the lungs *plus* universal adhesions of the lungs *plus* tuberculosis of the suprarenal glands *plus* tuberculous peritonitis (in a man, 21 years) 1

Extensive tuberculous infiltration of the axillary glands and glands in the neck *plus* small caseous foci in the lung *plus* miliary lung spread (in a male aborigine, three-quarters pure, 21 years) .. 1

Old fibro-caseous deep cervical glands on both sides *plus* laryngeal and pharyngeal tuberculosis *plus* caseous foci in the mediastinal glands *plus* secondary blood-seeding of the lungs 1

Scirrhous-looking tuberculous plaques on the pleura extending into the lung substance *plus* an old caseated focus at the left apex *plus* fibrosed tuberculous glands at the root of the lung and one in the lesser omentum. Death from fracture of the ribs and rupture of the liver and spleen (in a woman, 35 years) 1

Total 7

Slight or Semiquiescent Pulmonary or Thoracic Tuberculosis.

Deaths From Other Causes.

There were forty-eight persons who had died of other conditions, in whom slight or semiquiescent pulmonary or thoracic tuberculosis was present.

Small fibrous scarred areas, usually at the apex or apices, with small areas of caseation and occasional small cavities, the latter rarely larger than peas, and if larger with smooth, thin walls; a fibro-caseous patch five centimetres in extent at one apex (one case); an almond-sized caseated area at the apex or elsewhere (two cases); in occasional cases activity shown by the presence of a few miliary tubercles around a small cavity or caseated area 29

Ditto *plus* caseo-calcified area in a gland at the root of the lung 4

Ditto *plus* tuberculous pleurisy *plus* caseous specks in the mesentery 1

Fibroid area with cavity and some caseation.. 1

Scattered tuberculous foci in the lung 2

Soft or calcified nodules in a lower lobe 1

Caseo-calcified nodule 127 centimetres in diameter at the left apex 1

Patch of tuberculous broncho-pneumonia at one apex.. 1

Marble-sized area of conglomerate tubercles in a lower lobe 1

Patches of silicosis with small caseous areas (tubercle bacilli numerous) 1

Fibrosis and some caseation at both apices *plus* calcified mesenteric glands 1

A caseous focus at one apex or a cavity with some tubercles *plus* tuberculosis (calcified in one case) in a mediastinal gland 2

Caseous specks or caseo-calcification in a gland at the root of a lung 2

Fibro-caseous mediastinal gland 1

Total 48

Doubtful Examples of Tuberculosis.

The following have been separately carded as being doubtful cases. There were forty-three in all. In a number of instances the card has been marked merely as "possible tuberculosis" and a small number as "probable tuberculosis." I have come to the conclusion that a superficial thickening of the pleura at one or both apices is not necessarily indicative of an old tuberculous lesion, but is more in the nature of a thickening due to friction, as is so often seen in the capsule of the spleen.

A little thickening or scarring at one apex or a small, firm nodule without caseation or calcification (eight on the left and eight on the right) 16

Small scarred areas at both apices with or without adhesions 6

Thickening of the pleura at one or both apices not extending into the lung substance (probably not tuberculous) (two showing syphilitic aortitis) .. 9

Calcified streaks or plaques in thickened pleura at one or both apices 2

A few adhesions at the right apex 1

Small, firm adhesions at both apices 1

A small nodule at one apex and doubtful tubercles in a root gland of the opposite side 1

Small, fibrotic nodule at one apex *plus* a calcified nodule the size of a wheat grain in the spleen .. 1

Small, fibrotic nodule or deep scarring elsewhere in the lungs 3

Chronic pulmonary tuberculosis or silicosis at both apices with abscesses in an upper lobe 1

Mediastinal gland fibrous and hard, microscopically showing dense fibrosis and small areas of calcification 1

Head of femur absorbed, some mushrooming, no microscopic evidence of tuberculosis and history of alleged injury as a child 1

Total 43

THE DIFFERENTIAL ACTION OF X RAYS IN RELATION TO THE IMPROVEMENT OF RADIOTHERAPY.

By W. MOPPETT, M.B., Ch.M. (Sydney),
Cancer Research Biologist, University of Sydney.

A GREAT effort is being made throughout the world to unravel the mysteries concerned with the cause of malignant disease. In spite of the many interesting discoveries that have been made, it cannot be said that we have yet reached the stage when the results may be applied to the practical tasks of cure and prevention. The sufferer from malignant disease has at present the choice of two methods of treatment, surgery and radiation. Surgery is still to be regarded as the mainstay of treatment. Good results are obtained in early cases; often a mutilating operation only postpones a fatal issue; on rare occasions it appears to be accelerated. No doubt as time goes on, surgical technique will be continually improved and further statistical information will be gathered as to after results. Nevertheless, the horizon is limited, no great advances can be made, the only criterion of a successful result being to cut out every region which may have been permeated by malignant cells.

The future of radiation is decidedly hopeful. Occasionally an almost miraculous cure is produced, but apart from certain superficial conditions, such as rodent ulcers, the percentage of success is low and the results are notoriously uncertain. X rays act not so much by directly destroying malignant cells as by stimulating local tissue reactions. The resistance to radiation of malignant cells grown *in vitro* indicates that their direct destruction would be extremely difficult. Thus, one may regard a successful result as being produced through a cooperation with the natural defences of the body, a

principle which holds throughout medical practice. An even more important question is whether the autolytic products from the combined reaction of the malignant cells and normal tissues to X rays can call forth an immunity reaction. Is it possible, in other words, to produce a positive phase of immunity in the subject of malignant disease, so that one could be confident that any distant secondary deposit would be absorbed and a real and permanent cure effected? Lumsden has conclusively proved the existence of immunity in the case of inoculated tumours of rats and mice. He took an animal with two tumours and cured one by means of antisera or occasionally by non-specific destructive agents and in every successful case the other untreated tumour healed spontaneously and the animal proved to be immune to subsequent inoculation. His work points to the fact that malignant cells must be brought to a particular stage between life and death which may be termed the antigen stage, when they can call forth an immunity reaction. The case is more difficult in a spontaneous human tumour, but the rare complete cures of extensive inoperable growths indicate that some such process has been at work.[1]

Before considering how present methods may be improved, I shall outline briefly the properties of X rays and the extent to which these properties may be altered in an attempt to find the most suitable type of radiation for any particular clinical problem. X rays consist of electro-magnetic waves similar to light and extend over a wide range of wave lengths. In general the shorter wave lengths or hard rays have greatly penetrating properties and are suitable for treating deep conditions, while the longer wave lengths or soft rays are easily absorbed and may be used for superficial therapy. An ordinary X ray tube gives a fairly wide mixture of rays in which the short or long wave lengths may predominate according to the nature of the tube and the working conditions. In the application of X rays practically no attempt has been made to control the wave length used apart from the removal of very soft rays by means of a filter. The only condition that has been really investigated at present is that of dosage. The dose or total energy is a product of the intensity of the radiation multiplied by the time of exposure. Perhaps it would be better to express dose as total absorbed energy, since about 90% of the rays used in deep therapy pass right through the patient. Another way in which a dose of X rays may be altered, is by varying the time factor or distribution of the energy relative to time. For example, an erythema dose may be given in one short exposure or distributed over several days or weeks. These facts require further investigation and better measuring apparatus is needed. The primary difficulty with any X ray measuring device, such as a pastille, is that it absorbs only an unknown small percentage of the radiation incident upon it. Perhaps the ideal measuring apparatus would absorb the same fraction of the radiation used as the tumour to be treated. Nevertheless, with the mass of practical experience dosage does not explain the inconsistencies of radiotherapy and it seems reasonable to inquire into the precise significance of the other variable, namely the wave length.

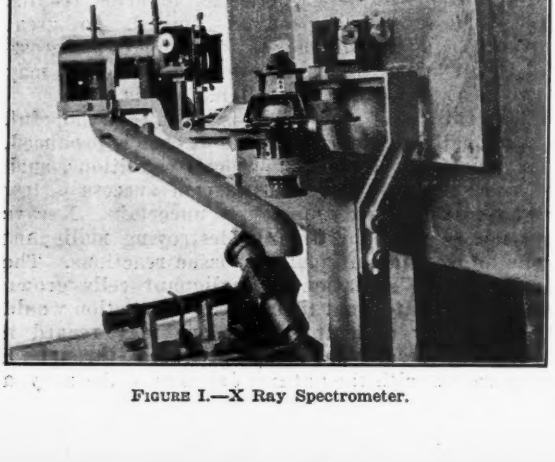


FIGURE I.—X Ray Spectrometer.

The main object of this paper is to describe a series of experiments devised to find out whether any particular wave length in the X ray spectrum might have special biological effects not shown by the adjacent wave lengths. If such a specific effect were found, in other words a differential action, many of the difficulties of X ray therapy might be explained. Up to the present time such an effect has been looked for experimentally by the use of various screens and spark gaps and the general opinion backed by theoretical considerations has been that no differential action exists. But the employment of screens and various spark gaps does not give radiation of one wave length, so that the question has not really been investigated. In attempting to solve the problem I have used an instrument known as the X ray spectrometer in which a beam of X rays may be split up into a spectrum similar to that obtained by a prism in the case of light. The apparatus is shown in Figure I. Mixed X rays direct from a tube pass through a small slit in the lead box and impinge on the crystal. The various monochromatic components or X rays of only one wave length are then reflected from the crystal at different angles. Moreover, the wave length of each portion may be calculated from these angles, a discovery that was made by Bragg. Thus, one has the means of investigating the biological action of known individual wave lengths in the X ray spectrum.

As a source of biological material I have used the chorio-allantoic membrane or breathing organ of the chick embryo at eight to nine days' incubation. This structure consists of two layers of epithelium enclosing a vascular mesenchyme which contains a variety of wandering cells and hæmatopoietic foci. It is closely applied to the shell or rather to the non-living fibrous shell membrane which lines the shell. The X ray spectrometer gives a small beam of X rays a few millimetres in cross section and this was allowed to fall on the allantois at a certain marked

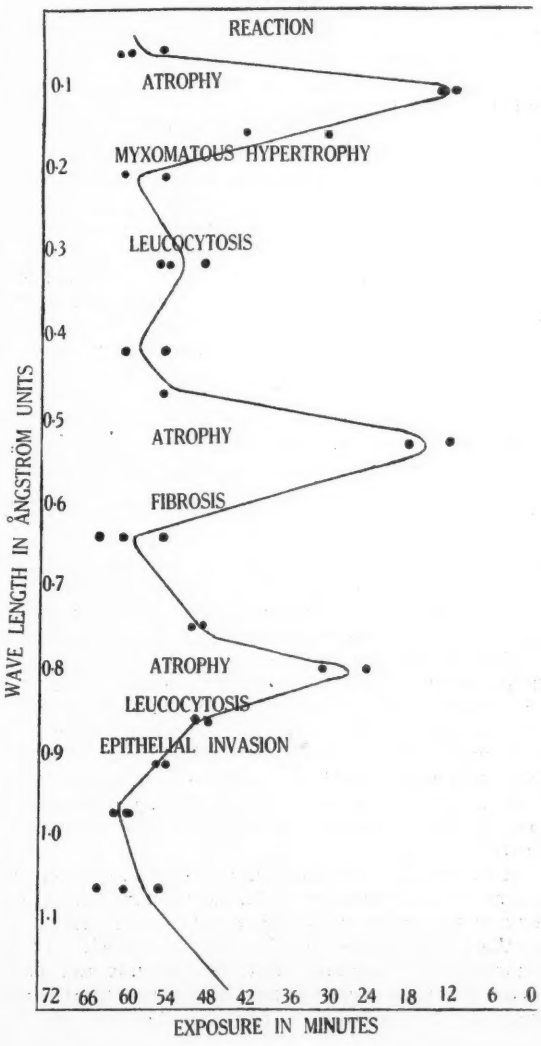
situation. To diminish scattering a small area of shell was sawn out and removed from this region, leaving only the fibrous shell membrane between the source of X rays and the structure to be observed.

A series of experiments was made in the above manner with different wave length X rays and the effect on the allantois observed. The immediate X ray effect is some chemical or physical change which is invisible, and like the latent image on a photographic plate cannot be detected as such. Therefore, the shell was closed after irradiation and the specimen incubated for a further four days. This was found a suitable time for tissue changes to become fully developed. In any search for differential action it is essential that for each wave length equal intensities and times of exposure should be given, otherwise a fallacy may arise because an apparent differential effect may be merely a question of dosage. It is quite possible to fulfill this condition if one utilizes what is known as the white radiation of an X ray tube, briefly that portion of the spectrum which contains no spectral lines. The intensity at each wave length was checked by an ionization method of measurement undertaken in cooperation with a physicist. This method is generally considered to be the best means of comparing the energy in different wave lengths. It depends on the property X rays have of rendering a gas a conductor of electricity.

A series of varying doses ranging from seventy-two down to six minutes were given on different adjacent parts of the allantois, so that the minimum dose required to produce an effect could be roughly estimated. When the irradiated specimens had been incubated a further four days, they were cut in half and the embryo and contents were poured out exposing the irradiated area *in situ*. A naked eye examination of the extent and character of the reaction, if any, was then made and the specimen was fixed for microscopic examination. The accompanying graph (Figure II) shows roughly the intensity of effect plotted against wave length and the character of each reaction is noted thereon. It will be observed that the X ray spectrum may be roughly divided into alternate regions of intense effect and minimal effect or more probably no effect since the small amount of scattered radiation which it is impossible to eliminate, would account for the minimal effects. Perhaps the most remarkable reaction is that described as atrophy. It was observed at three regions of the spectrum. A typical specimen is shown in Figure III. The region occupied by the beam of X rays appears almost as if punched out of the membrane, all visible structure is lost, the blood vessels *et cetera* ending in the raised edge which surrounds the reaction. Microscopical examination shows this area to be reduced to a thin amorphous band which has lost the property of taking stains (see Figures IV and V). This total destruction of tissues by a ray of specific wave length is a hitherto unsuspected effect of radiation. It is the more remarkable on account of the small amount of energy which is obtained by using an X ray spectrometer. The full dose of seventy-two minutes has, in fact, been sufficient only to blacken a photographic plate. According to the views which have hitherto been held as to X ray action this amount of energy would have no visible effect on living tissues of any kind.

It will be noticed in the graph that at certain other regions of the X ray spectrum situated in general at a slightly longer wave length than that which produces atrophy, the reaction is of a hypertrophic or inflammatory nature. Nevertheless, its extent is greatly in excess of the regions of minimal effect. These reactions may, therefore, be regarded as a characteristic response to certain wave lengths. Microscopical examination shows that the X rays of these specific wave lengths have a remarkable effect in selectively stimulating certain groups of tissues. Perhaps the most remarkable of these hypertrophic reactions is that described as epithelial invasion. It has been detected at only one region of the X ray spectrum as shown in the graph. A

FIGURE II.



microphotograph is reproduced in Figure VI. The epithelia are selectively stimulated and large masses of epithelial cells have separated off and invaded the mesenchyme producing the picture of a carcinoma. Another remarkable reaction is that described in the graph as leucocytosis which has been observed at two regions of the spectrum. The mesenchyme is crowded with leucocytes and immature blood cells and at a wave length of 0·86 Ångström unit these cells have invaded the epithelium, the reverse of the carcinoma like reaction. At another region of the graph a myxomatous reaction is noted, the epithelium was also thickened, but there was no sign of invasion. At another wave length there appears to be a definite tendency to stimulate the formation of fibrous tissue.(2)

I have already mentioned that the specialized energy which has produced these reactions, is very minute, much less than that used in an ordinary radiograph of a fracture. How can it be that such a minute dose can produce effects which at certain wave lengths amount to the rapid total destruction of living tissues. It might be supposed that the allantoic membrane is a very sensitive structure to radiation. Experiments in which a small area is exposed under similar conditions to mixed rays direct from the tube, indicate that the reverse is true. The allantois is quite resistant to radiation in the ordinary sense. Doses up to a thousand times that used in the X ray spectrometer are required to produce an inflammatory reaction. It appears from the above results that it is necessary to assume the existence of a phenomenon which I termed antagonism, that is certain wave lengths have the potential property of affecting living tissues, but if other wave lengths are simultaneously present, the action can be antagonized or nullified to most remarkable extent.

Many people hold the view that the biological action of X rays is due chiefly to secondary soft rays generated in the tissues and that the chief function of a beam of hard X rays is to carry the energy to a deep situation. Thus the precise wave length of the primary beam of X rays would be of no moment. The above experiments indicate that this theory is probably incorrect. When an effect appears sharply at a certain definite wave length it must be due to the wave length of a primary beam of X rays for the conditions of the above experiments have reduced scattering to a minimum and besides no one particular wave length that has been used, could be selectively effective in producing a rich supply of secondary soft radiation in the allantoic membrane. Rather it appears that the wave length and homogeneity of a primary beam of X rays is the all important factor in any effect it is desired to produce.

In estimating the value of the above facts on the future of radio-therapy it must be borne in mind that so far the results apply only to the allantois of the chick under the conditions stated. It is reasonable to suppose that this is not the only tissue to which such a remarkable differential effect applies. Rather from the fundamental character of X ray action which occurs in the first place on a single atom, does it seem probable that the results will prove general for all tissues or at least the laws will hold with some modification in detail. If such a condition really applies in the clinical application of radiation, it would mean that effects are produced only by a certain portion or rather by certain wave lengths of the radiation applied. Unnoticed variations in the voltage of the transformer would in many cases make a large variation in the amount of certain wave lengths present and this would account for many of the inconsistencies observed. This raises the question as to whether a practical source of homogeneous radiation exists. The ordinary methods of filtration do not give homogeneous rays and the X ray spectrometer cannot be used in practice owing to the small amount of energy available. A good output of homogeneous rays may be obtained by making use of the characteristic K radiations of certain elements chosen according to the wave length it is desired to produce. It seems probable that a more effective action might be obtained on a new growth by applying such wave lengths as produce atrophy in the egg experiments. It will be noted in the graph that atrophy was obtained at a wave length of approximately 0·1 Ångström unit. This particular radiation would be grouped with hard X rays; in other words it has sufficiently good penetrating properties to be used in deep therapy. It corresponds closely with the K radiations and absorption edge of the metal uranium. If these K radiations be applied to a tumour, a known wave length is utilized which cannot alter with fluctuations in the current supply since it depends on the element used, in the above example uranium. Experiments are in progress to determine whether the appplication of homogeneous radiation of known wave length will give more reliable and satisfactory results in the treatment of the subjects of malignant disease. Certain of the hypertrophic reactions suggest also that the wave lengths causing them might be effective in certain other clinical applications of X rays, such as the stimulation of contracting scars. The results give promise of a means which may at the very least greatly improve the percentage of success in X ray treatment. It is desirable that the matter should be thoroughly investigated.

It is possible that the application of the X ray spectrometer to biological purposes may throw some light on the more fundamental problem of causation. For example, a certain wave length actually causes epithelial cells to separate and migrate into the mesenchyme, just what happens in the most malignant forms of carcinoma. The balance or controlling force by which epithelial and mesenchymal cells remain in equilibrium, has been upset by the application of this particular wave length. But in the case of the specimen in which invasion of the epithelium by leucocytes is seen, the balance has also been upset by a different wave length and the "carcinoma effect" has been reversed. It may also be mentioned that many of the phenomena of inflammation appear separately and distinctly as

functions of different wave lengths probably associated with the disturbance of certain definite key atoms in the chemical economy of the cell. Whatever the future may be, many interesting problems are revealed in the unexpected results obtained by exposing living tissues to X rays of one known wave length.

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SECTION V.—PREVENTIVE MEDICINE.

THE POSSIBILITY OF FORECASTING OUTBREAKS OF DIPHTHERIA.

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IN these days it is well recognized that many individuals are immune to diphtheria, in spite of the fact that they may never have exhibited any symptoms of the disease. The presence of natural diphtheria antitoxin in the blood is one of the chief causes of this immunity. Diphtheria antitoxin is absent from the blood of those persons who are susceptible to symptomatic diphtheria. The absence of antitoxin in anyone's blood can be demonstrated by injecting dilute diphtheria toxin into the skin and observing a red patch appear after a varying time at the site of the puncture. Should no redness appear, there is natural diphtheria antitoxin in the blood of the tested subject, which has neutralized injected toxin. This simple procedure which is the rationale of Schick's reaction, makes it easy to determine whether or not any individual can contract clinical diphtheria when he is exposed to ordinary doses of the specific organisms. The latter qualification is necessary, because probably no one is completely immune or completely susceptible.

Under extraordinary circumstances the ordinarily immune person may have his resistance broken down by massive doses of diphtheria bacilli; undoubtedly many diphtheria bacilli are required to produce symptoms of diphtheria in even the most susceptible of subjects. However, immune and susceptible are convenient terms to use, provided that it is always understood that they mean a degree of resistance too high or not high enough to prevent symptomatic infection by an invading parasite under the natural conditions prevailing in the host's environment.

Bearing in mind the above limitations, individuals may be classed as immune Schick "negative" reactors or susceptible Schick "positive" reactors. In just the same manner a group of people can be considered as immune or susceptible to the spread of epidemic disease according as the percentage of immunes in the group is above or below a certain figure.

In the case of diphtheria it is obvious that the magnitude of an epidemic must depend to a great extent on the number of susceptible individuals in the population concerned. Moreover, it is obvious the ease with which bacterial parasites can get from one susceptible host to another, must largely depend on the density (number per unit or area) of susceptibles in the environment. The number of susceptibles in any environment can be obtained by finding out the number of Schick "positive" reactors in a fair sample of the population under examination.

Farr first pointed out the importance of density of population in the spread of epidemic diseases. As knowledge increased it began to be recognized that the density of susceptibles in an area was of greater importance than the total number of people per unit of area. But many years' study of disease outbreaks have convinced me that the immunes in a population cannot be ignored in analysing the spread of parasitic infection, which is found to depend not only on the actual density of the susceptible hosts in an area, but also on the ratio which exists between them and the immune hosts in the same area.

Besides supplying the actual number of susceptibles in a population, the Schick "positive" percentage also gives the relative number of immunes to each susceptible. For example, if there are 50% of susceptibles in one area and 25% in another of the same size, the density of susceptibles in the first area is twice that of the second. However, in the second area there are three times as many immunes to each susceptible as there are in the area which contains 50% of susceptibles. This means that in a 50% susceptible group a selected individual on the average will make one contact with a susceptible for each contact he makes with an immune; whereas in a community containing 25% of susceptibles there would be three immune contacts to each susceptible contact.

Similarly immune contacts would occur sixteen times as frequently in a group which contained 20% of susceptibles as in a group containing 80%, although the density of susceptibles in the latter group is only four times that of the former. If this dilution of the susceptibles by the immunes is an important factor in limiting the spread of bacterial infection, a population that contained fifty susceptibles and fifty immunes should, other things being equal, produce a greater number of cases of disease than a population containing fifty susceptibles and one hundred and fifty immunes. Because, although the density of susceptibles is identical in both groups and although the total density of the population is twice as great in the second group, yet the greater number of immunes in the second group will prevent the susceptibles from coming into contact with each other as freely as they would in the first group. Probably this diluting effect of the immunes on the susceptibles of a population is under certain conditions of even more importance than the actual density of susceptibles in determining the course of an epidemic.

In this short discussion of the subject I have purposely left out any consideration of carrier infection

which greatly complicates, but does not affect the main theme of the argument. If for susceptible, "infectable" is substituted, the argument will be seen to hold good.

The expression herd-immunity was used by Topley[1] to express the resistance of a population to the spread of bacterial infection. Herd-immunity undoubtedly depends largely on the percentage of susceptibles in a population. But it should be carefully noted, other things such as area being equal, that the percentage figure only gives the relative density of susceptibles, whereas the ratio of immunes to susceptibles gives the relative number of immune contacts in different areas.

Figure I. renders the distinction between relative density of immunes and relative number of immune contacts more easy to envisage. The straight diagonal line represents the relative density of immunes in a population occupying the same area. The curved line gives the ratio of immunes to susceptibles, which is equivalent to the relative number of immune contacts at the different percentages, that are indicated at the base of the diagram. The density is for convenience made unity when the immunes and susceptibles are equal in number, that is when the ratio is also unity.

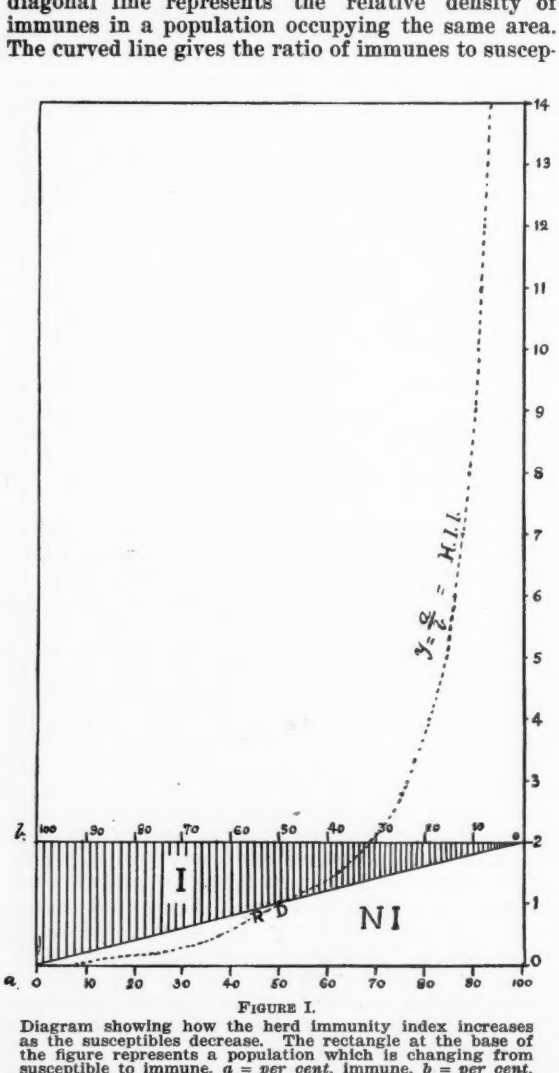

FIGURE I.
Diagram showing how the herd immunity index increases as the susceptibles decrease. The rectangle at the base of the figure represents a population which is changing from susceptible to immune. *a* = *per cent.* immune, *b* = *per cent.* susceptible, H.I.I. = herd immunity index, I. = infectable, N.I. = non-infectable, abscissa = relative density and herd immunity index.

The vertical figures give the relative densities and relative number of immune contacts in groups which contain different percentages of immunes. The resistance to the spread of an epidemic seems to increase much more in proportion to the immune-susceptible ratio than to the density of immunes, so for convenience the ratio of immunes to susceptibles may be called the "herd-immunity index." Figure I. shows how once the number of immunes form more than half of the population, a small increase in the immune percentage means a large increase in the herd-immunity index. Should all other factors in the spread of infection be equal, then the resistance to epidemic disease becomes proportional to the herd-immunity index, so that when the percentage of immunes is over seventy-five, a very small increase in percentage may produce a large increment in herd-immunity. Thus the herd-immunity index is especially significant to this paper, because at Greenwich, where most of my observations on diphtheria were made, the percentage of Schick "negative" immune reactions in different samples was always over fifty and sometimes over ninety. These two percentages correspond to a relative variation between 1·0 and 10·0 in the herd-immunity index, between 1·0 and 1·8 in the relative density of immunes and 1·0 and 0·2 in the relative density of susceptibles (see Figure I.). Therefore the most rapidly changing of these factors at high percentages is the dilution of susceptibles by immunes, as represented by the herd-immunity index. The study of diphtheria outbreaks at the Royal Naval School, Greenwich, made it seem probable that when herd-immunity increased beyond a certain point, the conditions became unsuitable for the spread of symptomatic diphtheria, though the environment remained suitable for the production of carrier infections. The following example will illustrate this point. At the Naval School during a period when the herd-immunity index was about four, the carrier rate for virulent diphtheria was on the average about 6%. During this time about twenty virulent cultures of diphtheria bacilli were isolated from apparently healthy boys and during the same period there were about two hundred Schick "positive" reactors in the school, yet, in spite of this prevalence of both virulent parasites and susceptible hosts, only one mild case of clinical diphtheria was seen.

The following hypothesis can now be stated. It should be possible by estimating the number of Schick "positive" reactors to get an idea of a population's degree of resistance to the spread of diphtheria and to find out by observations what value the herd-immunity index must exceed in order to render a community safe from a serious epidemic, even though susceptible Schick "positive" reactors are freely mixing with carriers of virulent diphtheria bacilli.

The degree of herd-immunity (value of herd-immunity index) which is necessary to render a population group safe from an epidemic, will vary in different environments, for instance, other things being equal, the herd-immunity index which is required to prevent the spread of infection, will have to be greater in ships, where men sleep in hammocks, often less than eighteen inches apart, than in those institutions where separate cubicles are provided. The lowest safe degree of herd-immunity must, unfortunately, vary with every complex of host and parasite. It will probably vary to some extent even with different strains of diphtheria bacilli, but as far as present knowledge goes it is not unlikely that with the same types of population inhabiting similar environments the herd-immunity index at which the spread of epidemic diphtheria becomes difficult, would vary by only a small amount from one year to another. Therefore should it be granted that it is possible to find out, by observations extending over a sufficient period of time, the minimum percentage of susceptibles in a certain community that must be present before symptomatic diphtheria can spread, then by simply estimating the percentage of susceptibles in the community it will be possible to state whether or not a diphtheria epidemic is to be feared.

In Greenwich Naval School a percentage of about thirty susceptibles seemed to be necessary before a big outbreak of diphtheria was possible. At the termination of one epidemic the percentage of susceptibles in this school was reduced to fifteen. Therefore the school was safe from diphtheria, until such time as the introduction of susceptible recruits again reduced the herd-immunity to a dangerous degree. Suppose after this epidemic no further change took place in the immunity of the boys living in this school or of those entering the school, the herd-immunity would then decrease term by term, because the recruits who joined the school, would contain a greater number of susceptibles than the boys who left the school each term. Hence it is possible to go further than merely stating that the herd-immunity is such that no diphtheria outbreak is to be feared at present, but, by Schick testing the new entries to a school to calculate the minimum time which must elapse in order that the susceptible recruits may reduce the immunity to a dangerous value.

For example in a school such as that at Greenwich the total population remained constant at one thousand boys. On the average one hundred boys joined each term to replace the hundred boys who had completed their schooling. The new entries contained on the average 50% of susceptibles. Hence fifty susceptible boys were added to a population of a thousand every four months, which increased the total non-immunes in the school by 5% per term. If there are only 15% of boys who are not immune to diphtheria to start with, and if there are no susceptible boys leaving the school, it will require three batches of recruits to increase the susceptible population from 15% to 30%. That is to say, if at least 30% of Schick "positive" reactors are required to render the conditions suitable for an outbreak of diphtheria, the school is safe for at least eight months, which is the time elapsing between the entry of the first and third batch of fifty susceptible boys.

Theoretically it should be possible to forecast approximately the actual time rather than the minimum time that the herd-immunity will take to sink to a dangerous level. The actual time taken will always be greater than the minimum time as calculated above, because a certain number of susceptibles will be present among those leaving the school each term. Also, under certain conditions, many of the Schick "positive" reactors will require immunity under the influence of the school environment.

Figure II. demonstrates how herd-immunity to diphtheria increased with length of residence in the Royal Naval School. The columns represent the percentages of Schick "negative" immune reactors in groups of boys, who are arranged in order of their school seniority. The figure shows that the senior boys possessed at the time of testing a much greater degree of herd-immunity than the new entries. The resistance to the spread of diphtheria among the same groups of boys is better envisaged by Figure III., which gives the ratio of immunes to susceptibles, that is the herd-immunity indexes, arranged in the same order as in Figure II.

The change in the Schick reaction from "positive" to "negative" is undoubtedly due to the inhalation of doses of diphtheria bacilli or their products that are contained in the droplets which are sprayed into the air by patients or carriers of virulent diphtheria

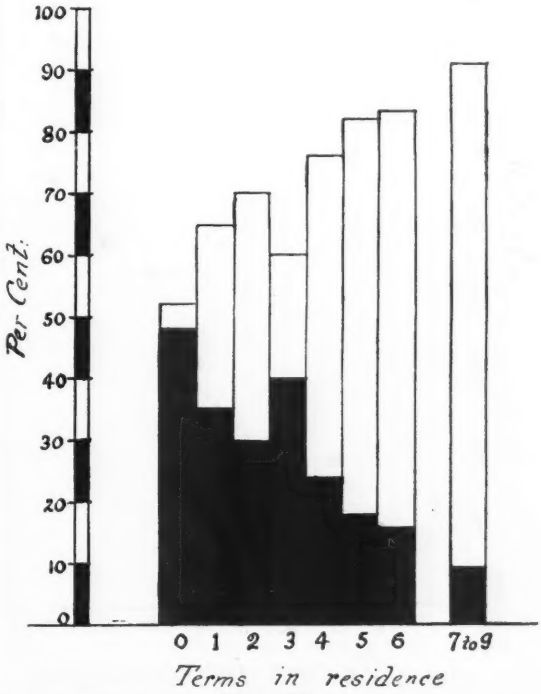

FIGURE II.
Effect of length of residence on herd immunity to diphtheria. Schick tests, Royal Naval School, March, 1924. Black columns = susceptible, total columns = *per cent.* immune.

bacilli. The reasons for this statement have been fully discussed elsewhere.[2] The rate at which a school increases its herd-immunity may be measured by the percentage of Schick "positive" reactors which become "negative" term by term. Figure IV. shows how this rate of immunization is correlated with the presence of symptomatic diphtheria. In the figure the dotted line gives the average rate of immunization as measured by the Schick reactions of the same sample of boys who were tested on the dates corresponding to the points of reference joined by the dotted line. In Figure IV. the dotted line is superimposed on a histogram of the morbidity from diphtheria term by term which makes it easy to see how the rate of immunization follows the incidence of diphtheria.

Therefore herd-immunity tends to increase rapidly in the presence of an epidemic of diphtheria. It will also increase though more slowly in inter-epidemic periods, provided that carriers of virulent diphtheria bacilli are present in the environment. For practical purposes Schick immunity once acquired may be considered permanent. Therefore in estimations on the rate of change of herd-immunity there is no need to make allowance for Schick "negative" reactors becoming "positive" again.

When the minimum time taken for the susceptible population of a community to increase from 15% to 30% was calculated, it was assumed that all the "positive" reactors who entered the school remained positive and that all the boys who left the school were "negative" immune reactors. Actually, as just explained, many Schick "positive" reactors became "negative" at varying intervals after joining the school and of course a few "positive" reactors were always present among the boys who left the school at the end of each term.

For the sake of illustration, let it be supposed that in a community whose numbers remain constant at one thousand, every four months fifty susceptibles join and ten leave. Then the increase in susceptibles will number forty, or 4% of the total population per period of four months. Hence it would take 3·75 periods or over a year for the percentage of susceptibles in the community to increase from fifteen to thirty. If in addition twenty of the susceptibles already in the community, gain immunity every four months, then the total increase in susceptibles would be only 2% instead of 4% per term and it would take 7·5 periods of four months or 2·5 years before the herd-immunity index of this imaginary community would be reduced from about six to two, that is the percentage of susceptibles increased from fourteen to thirty-three.

The discussion so far has been concerned only with communities which, having acquired a safe

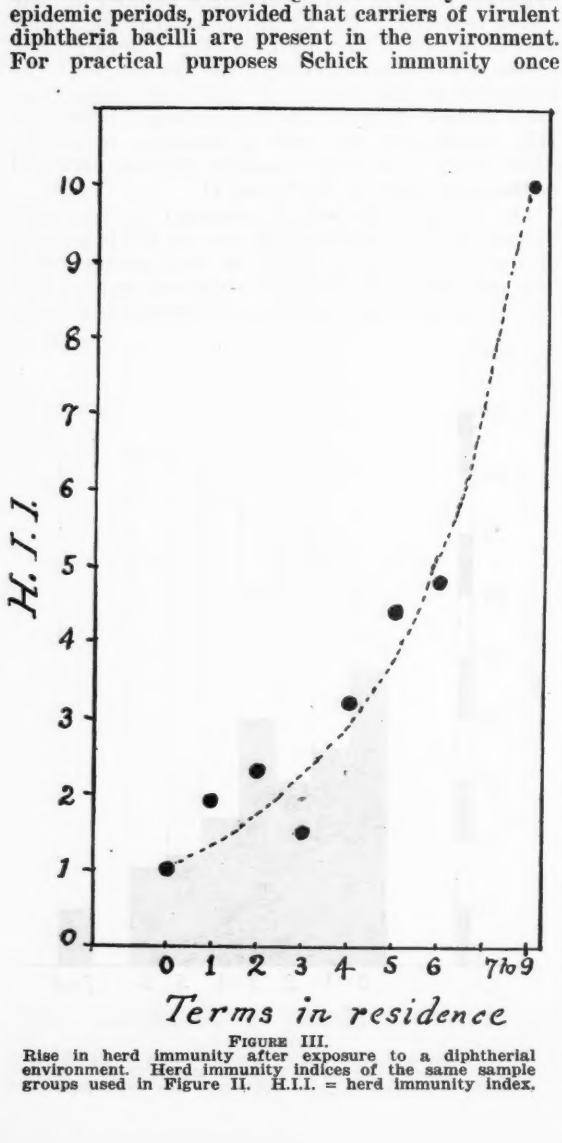

FIGURE III.
Rise in herd immunity after exposure to a diphtherial environment. Herd immunity indices of the same sample groups used in Figure II. H.I.I. = herd immunity index.

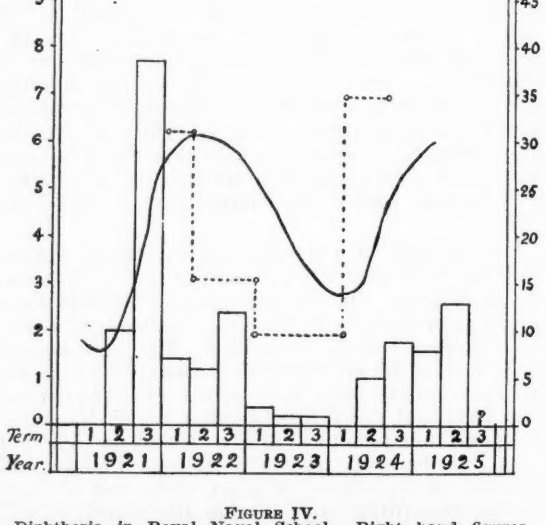

FIGURE IV.
Diphtheria in Royal Naval School. Right hand figures = *per cent.* attacked per term and herd immunity index. Left hand figures = average *per cent.* positive Schick reactors becoming non-susceptible per term. Columns = incidence of symptomatic diphtheria, dotted line = average rate of immunization per term, curve = approximate variation in herd immunity index.

degree of resistance to bacterial spread, are losing it. The possession of a high degree of resistance to infection implies that the population has at some time or another been exposed to an intense diphtherial stimulus, such as occurs during an epidemic. An increasing herd-immunity necessitates that the number of susceptibles who acquire immunity each period, should be greater than the difference between the number of susceptibles who enter, and the number who leave the environment each period. In the example given above the decrease in immunes due to the change of population was 4% per term. This decrease would be just counterbalanced if the rate of immunization due to the school environment was also 4%. In such a case the degree of herd resistance would be maintained at a constant level. Should the rate of immunization rise above 4% to 9%, the herd-immunity would then increase by 5% per four monthly period. An increase as rapid as this appears to occur only during an epidemic of diphtheria. If such data as the above were available, they could be used to foretell the end of epidemics. For example, given that in a certain population diphtheria could not spread once the percentage of immunity reached eighty and given that the rate of immunization was constant at 5% per four months, then, if at a certain time during the progress of an epidemic the immunes formed 70% of the total population, it would be possible to prophesy the epidemic would cease within eight months.

If the foregoing propositions are correct, an important corollary follows. Should Schick examinations show that herd-immunity is increasing in a community, in which clinical diphtheria is absent, but that more susceptibles join than leave the community, then two things can be inferred; first that carriers of virulent bacilli are present, because diphtherial proteins are needed to produce the known increase in antitoxin immunity; second the herd-immunity must be above the value at which symptomatic diphtheria is able to spread or otherwise the virulent bacilli in the neighbourhood would have caused cases of diphtheria. Also since the herd-immunity is already above the danger point and is still increasing, there need be no immediate fear of a diphtheria outbreak.

The observations on diphtheria which were made at Greenwich, showed that at the end of 1922 after a big epidemic had ceased, the herd-immunity index of the school had reached the high figure of six. No diphtheria of any consequence occurred during 1923 and early in 1924 the herd-immunity was found to have dropped to four. In June, 1924, the herd-immunity index was still on the decrease (see Figure IV.); also about this time experimental throat swabbings showed that, though no cases of symptomatic diphtheria had been seen, yet more than 5% of the boys who were bacteriologically examined, were found to be carriers of virulent diphtheria bacilli. The falling herd-immunity combined with the presence of virulent carriers caused me to warn the school authorities that another outbreak of diphtheria was imminent. The immunization of the Schick "positive" boys with toxin-antitoxin mixture was also advised. But as Figure IV. shows the expected epidemic broke out in July, before there was time to organize the advised prophylactic inoculations. Thus an epidemic of diphtheria was definitely foretold on the principles I have tried to explain in this paper. Even if this forecast was in the nature of a fluke, it does not affect the argument. The Greenwich work had not been designed for the purpose of forecasting epidemics, but the analysis of the numerous observations that had been made during previous years, suggested that the forecasting of epidemics and "herd-prognosis" might soon become a practical proposition. There should be no technical difficulty in a school, such as that at Greenwich, in Schick testing each term all the new entries, together with all those boys in the school who had given positive results the term before. Such a routine would supply all the information necessary to calculate the degree of herd-immunity and its rate of change term by term.

To summarize, when the percentage of immunes is above a certain value which will carry with every environment, and every complex of host and parasite, the resistance of the herd to the spread of symptomatic infection becomes so high that epidemics are unable to start or continue, even when many susceptible hosts and infecting parasites remain in the environment. In the case of diphtheria this degree of herd-immunity can probably be ascertained for certain population groups. Once ascertained Schick testing will show:

1. When an epidemic is to be feared;

2. When an epidemic of diphtheria in actual progress may be expected to cease;

3. How many individuals in the community must be rendered immune in order to raise the resistance of the herd to a safe value.

The interest of this paper is perhaps more academic than practical, because public health authorities should not waste time attempting to foretell diphtheria outbreaks, but rather they should spend their energies in insuring that no epidemics can occur, by immunizing all Schick "positive" reactors with toxin-antitoxin mixtures. However, should it prove a fact that in order to render a group, as distinct from an individual, immune to diphtheria, it is not necessary to remove the power to react to the Schick test of all the inoculated susceptibles, then a point of great practical importance does emerge from this work. The present prophylactic technique means, first a Schick test, followed by three inoculations and a subsequent Schick test. Moreover, if the result of the second test is positive, as it is in about 20% of cases, further inoculations and tests have to be done. Therefore the victims of diphtheria prophylaxis have to undergo at least seven punctures and perhaps many more. This repeated needling scares young children and worries their parents and teachers who not without some reason begin to wonder if the risk of diphtheria is not preferable to its prevention. The time, anxiety and expense to everyone concerned

in prophylactic inoculation could be halved, if it was found that two or even one injection of toxin-antitoxin mixture would raise the herd-immunity to a safe level, because then one or two of the inoculations and the second Schick test could be dispensed with and the seven or more punctures reduced to three or four. Under such a simplified technique many Schick reactions would remain "positive" after inoculation, but Glenny, Südmersen and Allen's[3] work, together with certain as yet unpublished observations made at Greenwich, make it not unlikely that the inoculated diphtheria prophylactic would act as "a primary stimulus" which would sensitize the subject so that when a "secondary stimulus" in the shape of an ordinary dangerous dose of diphtheria bacilli was received, the response would be a rapid increase of antitoxin immunity, rather than an attack of clinical diphtheria. If future work shows that such is the case, diphtheria immunization will become much more popular with both the doctors and the public.

In conclusion and in order to forestall criticism, it must be pointed out that this paper lays no claim to prove that epidemics of diphtheria can be accurately foretold, but it indicates how in the not so distant future public health authorities will be able to calculate the probabilities of danger from certain infections and being thus forewarned, they will be better able to organize measures of prevention. This thesis also serves a useful purpose, if it emphasizes the necessity of treating all the phenomena of infectious disease as the interactions of three variables, the host, the parasite and the environment and shows how futile it is to draw any conclusion from a change in one of these variables if nothing is known of the other two on which the first depends. Also the effect of motion and time must ever be borne in mind, since the dissemination of parasites is a problem in dynamics not statics.

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(3) A. T. Glenny and H. J. Südmersen: "Notes on the Production of Immunity to Diphtheria Toxin," *Journal of Hygiene*, 1921-1922, Volume XX., page 176.

(4) A. T. Glenny and K. Allen: "Active Immunity to Diphtheria in the Absence of Detectable Antitoxin," *Journal of Hygiene*, 1922-1923, Volume XXI., page 100.

DR. T. MCKIBBIN (Wellington) held that it was of great value to get an odd immune person among a community. To carry out the Schick test uniformly through a community was a difficult matter, but was a possibility in schools and institutions. It was possible to predict diphtheria epidemics by seasonal incidence alone. Epidemiological waves were evident in diphtheria and for the last several years diphtheria had been on the wane. The last high death figure for diphtheria in New Zealand had been in 1917; since then the death rate had receded. Probably this was only a phase and sooner or later there would again be a flare up. Predictions might be made along the lines indicated by Dr. Dudley, but might also be made from the weekly incidence of diphtheria for years plotted on paper. When the incidence and death rate rose sharply and continued for two or three weeks, an epidemic might be foretold. Enteric fever and scarlet fever were also decreasing, but this, too, was probably a phase only. Other diseases, for example, meningitis, were on the increase. It seemed as if a change must come in the infectious disease outlook. Influenza was a definite problem. Minor epidemics arose, for example in 1920, a year of influenza, whooping cough and measles, in 1926 whooping cough, mumps, measles, influenza, increased broncho-pneumonia, bronchitis. The checking of influenza was difficult. Notification had had to be curtailed to the virulent type only, such as the pneumonic and the septicæmic types. Acute primary pneumonia as a notifying name was useless. Epidemic influenza was more of the broncho-pneumonic type. It would be of benefit if the registrar of the district were to notify the Health Department of every case of pneumonia, bronchitis and influenza which occurred. At present there was no real dividing line between these three as notified by the practitioner.

DR. H. O. LETHBRIDGE (Narrandera) spoke as a practitioner advocating the value of the direct smear in diagnosis. The disadvantages were the loss of time and unreliability, but in practice a direct smear made from every sore throat led to a classification into three classes: (i.) those in which no pathogenic bacteria were seen, (ii.) those in which the Klebs-Löffler bacillus was recognized and (iii.) those in which bacilli, spirochætes and other organisms were present in conjunction with one another. For the second class isolation of contacts and immediate treatment of the patient were indicated. The third group was difficult, but a conclusion could be arrived at. In an epidemic in a small Australian town he had collected about 900 direct swabs. The result had been a great decrease in diphtheria admission as the children from whose fauces diphtheria bacilli had not been grown, had been excluded. Schick testing was impracticable in general practice and toxin-antitoxin treatment was looked on with disfavour by the public. He held that direct examinations were of great value.

DR. BAKER-MCLAGLAN (Wellington) thought that the punctures were a drawback in school work. It would be better if the number of punctures in immunization could be reduced.

DR. HARVEY SUTTON (Sydney) said that Commander Dudley had formulated good reasons why epidemics rose and died down again. The seasonal incidence was marked in Australia as everywhere in the world. Cycles in infectious disease had not yet been explained, for example whooping cough five-yearly interval, mumps six-yearly interval and then a pandemic. Something in the organism itself must control the cycle factor. The decrease in diphtheria incidence was due partly to decreased birth rate. With increasing families the number of susceptibles increased. He thought that Schick testing was enhanced in value by omitting the preliminary Schick test in young children of, say, five to ten years and proceeding to immunize the whole of the group. Even the partial immunization, for example, two doses, would be effective as Dr. Dudley had pointed out. Contact of children in school automatically increased the number of immune. There were more susceptibles among country children than among city children.

PROFESSOR C. E. HERCUS (Dunedin) agreed that age dilution (the declining birth rate) was one of the explanations of decreasing diphtheria epidemics. In cerebrospinal fever in the war when the percentage of carriers reached a certain warning level an epidemic was ahead. Dr. Dudley's finding for diphtheria was similar and was full of significance. He felt with Dr. Sutton that it should be a routine to immunize all school entrants. The treatment of toxin by formaldehyde gave a maximum antigenic power with no toxicity. Tetanus toxoid was recommended as a routine prophylaxis. Similarly diphtheria toxoid would shortly be used and would prove more effective than the previous toxin-antitoxin mixture. In future all children at commencement of school life should be immunized. There would then be no need to worry about the later school ages. When the primary stimulus had been given, contact with massive infection resulted in a great antitoxic

response. Tissue education was complete and the subject did not contract diphtheria. Simple primary stimulus by toxoid would solve the problem of future diphtheria epidemics. He paid a tribute to Dr. Lethbridge's interest in public health activity. The direct smear was certainly of great importance; general experience was in favour of culturing, but the former helped in the differential diagnosis.

SECTION VI.—OPHTHALMOLOGY.

A NOTE ON THE PATHOLOGICAL EFFECTS OF PENETRATING WOUNDS OF THE EYEBALL.

By A. J. HALL, M.B. (New Zealand),
Dunedin.

THE late Mr. Hislop Manson, of Glasgow Eye Infirmary, whose sudden death in the operating theatre about twelve months ago removed at an early age a very prominent and much esteemed ophthalmologist, was kind enough to agree to let me have pathological findings in a series of cases of penetrating wounds of the eyeball, for which class of injury his clinic, owing to its proximity to numerous iron-works, was peculiarly well placed.

His associate, John Marshall, of the Pathological Department of the Eye Infirmary, knowing about this promise, took on at Manson's death the duty of fulfilling the promise and the series of stereophotographs which I am showing, are entirely his work.

I must, therefore, disclaim any important part in this matter as it very largely represents his own findings and deductions.

I have from Marshall's laboratory a series of about thirty-five cases represented by half photographs, stereoscopic photographs and microscope slides. These demonstrate that infection following penetrating wounds may produce: (i) Panophthalmitis, (ii) vitreous abscess or posterior endophthalmitis, (iii) anterior endophthalmitis, (iv) plastic iridocyclitis, (v) proliferative fibrosis arising from the cut tissues at the wound.

The first group, panophthalmitis, is one which all of us will have cause sadly to recall in our operative experience, however diligent we have been in our efforts to sterilize.

In the very acute types within a few hours after operation or injury the patient developes violent pain and intense inflammation of the whole eyeball (see Figures I, II and III). The signs are intense conjunctivitis, early chemosis, a muddy or yellow iris, yellow reflex from the pupil if the condition of the cornea and anterior chamber has not had time to obscure the pupil. Œdema of the lids with increase of pain and protrusion and immobility of the globe ensue and relief is obtained only by evacuation, spontaneous or artificial.

The outstanding pathological signs are a thick layer of purulent exudate on the iris, ciliary body and retina, but very little reaction in the chorioid which appears to be protected by the retina or by the lamina of Bruch. There may be a round-celled infiltration, but polymorphonuclear leucocytes in the chorioid are rare. In these cases there is no sign of organization of the exudate.

The second class, vitreous abscess or posterior endophthalmitis, which may be regarded as an intermediate type between the frankly purulent panophthalmitis and the non-purulent but plastic iridocyclitis types, is represented in Figures IV, V, VI and VII. These cases are less virulent than the first. The onset is not quite so violent, there is little pain and the eye which looks as if panophthalmitis were about to develope in it with its accompanying pain and inflammation, after a few days shows signs of improvement. But this semi-acute stage does not pass off and the "irritable" globe gradually passes into the condition of the "shrinking" globe. In some cases the initial signs are quite mild, but shrinking results after a tedious stage of prolonged irritability.

If the injury has not resulted in any damage to the transparency of the lens, the yellow reflex in the pupil soon becomes evident. In some cases there may be very little alteration in the iris.

The characteristic feature in this class is that the inflammatory reaction is greatest in the ciliary body. A dense mass of purulent exudate is thrown out into the vitreous from the purulent cyclitis, causing the yellow reflex and the contraction of this mass separates it from the retina.

There is no purulent retinitis as in the first class, but there is a definite round cell reaction in that membrane more intense in the inner layers and masses of round cells are thrown out towards the vitreous, so that eventually a pseudo-membrane (suggestive of a hyaloid membrane) may be formed (see Figure VII).

Proliferation changes occur in the ciliary body and the "abscess" may become enclosed in fibrous tissue. The gap between this mass and the retina is soon obliterated by the latter detaching and following the vitreous mass (see Figure V). Fibrosis and shrinkage occur later if the eye is left in the head (see Figure VI).

There is a mid-class of *endophthalmitis septica anterior* in which an infection of a wound in the posterior parts of the eye extends forward and causes septic iritis with hypopion or turbidity of the aqueous, with or without vitreous abscess.

These are difficult cases in which to form a prognosis. The vitreous cannot be seen owing to the turbid aqueous and all depends on the presence or absence of a vitreous abscess as to whether excision is required or not. The test by perception of light and perhaps by reaction of pupil to light should be carried out.

The fourth consequence of perforating wounds is the class of septic plastic irido-cyclitis. Clinically these are not easily separated from some cases of the previous class. The eyes are strongly injected, but there is no chemosis. The pain is not so acute, but may be more or less severe. The iris is discoloured, but not yellowish-green as in purulent cases. Rarely is there hypopion. The anterior chamber is deep. There is the greatest difficulty in keeping the pupil wide, owing to the plastic quality. The appearance of these eyes is seen in

Figure VIII, but in some cases it may resemble the vitreous abscess type.

The exudate into the vitreous in this class is cobwebby and much less dense. It is made up of round cells and fibrin and is not a polymorphonuclear mass as in the other types. It is a plastic product. Characteristically the ciliary body and iris show early proliferation of their connective tissue elements and adhesions form.

In all the cases there is infiltration with round cells of the inner retinal layers but less so than in the second class. Newly formed tissue may thus be found from the part of the ciliary body near the *ora serrata* at the point where the vitreous fibres are attached. This may spread out into the vitreous towards the posterior pole of the lens or forward along the ciliary body and when it contracts, the retina may be pulled inwards towards the pole of the lens (see Figure IX) or up over the ciliary body (see Figure XII) and it may cause separation of the ciliary body (see Figures X and XI).

The fifth and last group consists of those cases in which there is a purely localized reaction with proliferation changes in the cut tissues of the wound. There may be some reaction of the plastic type on the ciliary body, but this produces much less alteration in the structure of the eye than the proliferation in from the wound.

Clinically these are somewhat like the fourth class, but very often the eye appears to do fairly well for a few days with little irritation, but its tension keeps low and when after a considerable time the media have cleared, the proliferation effects can be seen perhaps as a feathery band of white passing out on to the lens from the region of the wound. Wounds of ciliary body and sclera may produce this type, but wounds of the cornea may do so too, especially where there is some scaffolding along which the tissue can grow, such as blood clot, vitreous in the wound and so on.

Figures XIII and XIV show a band connecting retina to ciliary body.

I have purposely avoided the question of chemical and biochemical reactions from the retention of metallic bodies as I desired to deal with the more immediate effects of bacterial invasion rather than remote changes. I am afraid that from the clinical standpoint I have not been able to provide anything of great practical value but I feel very indebted to Dr. Marshall for the work he has sent and I feel sure that it will appeal to you all as a matter of much interest, in that it shows the manifold ways in which penetrating wounds may react pathologically under the provocation of infection whether it be derived exogenously or endogenously.

In regard to the question of precipitates of exudates on the back of the cornea in irido-cyclitis of non-purulent type, the pictures do not, of course, illustrate any of these features. They are characteristic of chronic cyclitis. But it might not be out of place to digress for a very short reference to these deposits.

Our knowledge of these has vastly improved with the use of the slit lamp. When a beam of light traverses the cornea, anterior chamber and lens, it is, of course, visible in the optical section of the cornea and of the lens, but it is invisible or almost so in the normal aqueous But any uveal inflammation may produce any degree of cloudiness or turbidity of the aqueous, so that the lamp beam becomes visible in crossing the chamber by reason of the particles.

In children with perfectly healthy eyes a few isolated cells (? white corpuscles) can be picked up with the Nitra lamp, but it requires expert focussing. These isolated non-pathological cells (if one may use such a term) differ from pathological ones by the fact that they never agglomerate.

Unpigmented precipitates on Descemet's membrane are seen at the onset of irido-cyclitis and sympathetic ophthalmitis with a high magnification, but in most cases the deposits are coloured.

But it is claimed that invariably an earlier sign still is that known as bedewing of the endothelium. This is an appearance of the cells as if they contained droplets and it is seen only in the zone of specular reflection (see Figure XV).

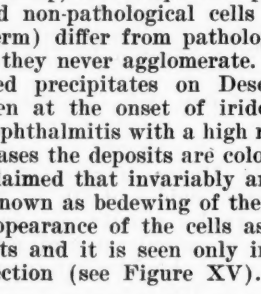

FIGURE XV.
Diagram to illustrate convection currents in the aqueous fluid (from Goulden's translation of Kobe's book).

Such a sign may be observed before any noticeable injection can be made out in the eye about to be excited and it is, therefore, of the highest value.

After an attack of irido-cyclitis has quietened down, resorption of deposits may take place, but often a certain amount of fine dust (fibrin fragments and so forth) remains, causing some slight haze or loss of lustre when the cornea is transilluminated.

The precipitates of the cellular output into the aqueous deposit in the usual triangular formation on Descemet's membrane, but not always. In some insidious cases of irido-cyclitis they are seen on the very centre of the cornea (Gouldon) and Vogt says this may disappear and return repeatedly before any injection of the globe appears.

It is interesting to consider why the precipitates should take up the well known triangular form of distribution on Descemet's membrane.

This may be explained by the thermic currents in the aqueous causing the precipitation as can be seen by the diagram taken from Goulden.

REPORT OF A SERIES OF CASES OF RETINITIS PIGMENTOSA OCCURRING IN ONE FAMILY.

By CYRIL DICKSON, M.B., B.S. (Melbourne)
AND
LEONARD J. C. MITCHELL, M.D., B.S. (Melbourne).

During 1926 three members of one family attending the Ophthalmological Out-Patient Department of the Melbourne Hospital were discovered to be

suffering from *retinitis pigmentosa* and evidence of disease in collaterals can be deduced.

The father, *ætatis* fifty-four years,, and mother, fifty-six years, had six sons and four daughters, of whom four sons and four daughters survive. Of the sons, M.R., *ætatis* thirty years, one of twins whose brother died in infancy, is normal; R.R., *ætatis* twenty-eight years, presents gross deafness. His vision is 6/18 in both right and left eyes with definite contraction of the fields of vision. Examination of the fundi reveals optic atrophy, vitreous opacities and well marked, typical changes of *retinitis pigmentosa.*

A.R., who died at twenty-one years, was blind and deaf. No details are available, but the inference is that he was suffering from the same disease.

The vision of B.R., *ætatis* nineteen years, in each eye is 6/6. The fundi are normal, and there is no contraction of the visual fields.

The vision of D.R., *ætatis* sixteen and a half years, in the right eye is 6/9 and in the left eye 6/12. Examination of the fundi reveals *retinitis pigmentosa* at the extreme periphery. The visual fields are very much contracted. The late Dr. Frank Andrew reported typical end organ deafness.

Of the daughters, U.R. and G.R., *ætatis* twenty-three years and twenty-one years respectively, are normal. K.R., *ætatis* fifteen years, has 6/9 vision in each eye. Definite changes of *retinitis pigmentosa* are present in both fundi together with evident contraction of the visual fields. "Typical gross end organ lesion" was reported by the late Dr. Andrew who examined the ears.

The vision of A.R., *ætatis* thirteen years, is 6/5 in each eye. The fundi are normal.

The father has been deaf since the age of twenty-nine years. No eye defect was detected on examination.

The mother is normal.

The father had two deaf and dumb cousins, both dead. This is of interest as the condition is reported to occur in 4% of deaf mutes. In addition he has four sisters and two brothers who are normal and whose children are stated to be normal.

There is no history of eye or ear disease among the mother's relatives.

The Wassermann test (blood) was applied throughout the family, but no reaction was obtained.

Ætiology.

The disease is better called pigmentary degeneration of the retina and runs a very chronic course. It attacks both eyes and developing in childhood is probably congenital in many cases, thought its discovery may be made some time after birth. Heredity plays a great part in its production, as does consanguinity. Occurring as it does in families, it affects females more than males and is often associated with other defects and malformations. Posterior polar cataract sometimes supervenes in later life.

Treatment.

Treatment is useless, mercury, potassium, strychnine, electrical stimulation being alike unavailing.

While blindness supervenes later in life in many cases, the patient should not be unduly depressed by a blunt prognosis, for even if a cataract developes it is worth while removing it to give some remnant of vision.

In the family under observation, two (and almost certainly three) males were affected and one female, the percentages being fifty and twenty-five respectively. The age or position in the family does not seem to be a determining factor.

DR. A. M. MORGAN (Adelaide) mentioned the history of two patients with *retinitis pigmentosa* in one family. Neither were able to talk properly, but hearing was perfectly normal in both. He mentioned another patient whose field of vision was not altered; there had been no alteration in the clinical appearance for over twenty years, the only trouble this patient had had was night blindness. No treatment had been used except Blaud's pills given occasionally. With reference to consanguinity being a factor in the ætiology, he mentioned a case in which the parents were first cousins. The eldest of the family of this marriage had *retinitis pigmentosa* and was stone deaf. The other members of the family had no retinitis, but all became very deaf, the deafness not coming on until about the age of twenty.

SIR JAMES BARRETT (Melbourne) said that he had had Wassermann tests carried out with the serum of all his patients. In the majority no reaction had been obtained. One of his patients whose sister had *retinitis pigmentosa* of a typical kind, had typical *retinitis pigmentosa* without pigmentation. In mentioning consanguinity as a cause he thought that medical men were inclined to put too much stress and attach too much importance to the bad effects following on the marriage of near relations. He pointed out that degeneracy had been found in the Pharoahs of the eighteenth dynasty. It had been the custom for the Pharoahs to marry their sisters. He mentioned that this period was the most notable period in Egyptian history. He warned medical men to be chary about advising against marriage of near relatives, for example, first cousins, till they had gone carefully into their history. If the history of both sides was good, there was no reason why they should not be married.

DR. A. J. HALL (Dunedin) said that he would like to know what degree of deafness was present in the patients referred to in Dr. Mitchell and Dr. Dickson's paper. In a patient whom he had shown, he was satisfied that deafness was of a catarrhal type. He thought speech defects were common and in the case mentioned the patient had not learned to speak until the age of seven. In this case also posterior capsular cataract had not developed until late in the disease. There had been no sign of cataract in 1913, but it had been present when he had reexamined the patient in 1926.

SQUAMOUS-CELLED CARCINOMA OF THE LIMBUS EXTENDING OVER THE CORNEA.

By Z. SCHWARTZ, M.B. (Melbourne), F.R.C.S.E., D.O.M.S. (London).

Clinical Assistant, Melbourne Hospital and Eye and Ear Hospital (Melbourne).

The patient was a woman forty-three years of age, machinist by occupation.

History.

When first seen on December 22, 1925, her history was that two years ago she attended a hospital complaining of the left eye.

In February, 1924, her vision was recorded at that hospital as 6/5 in the right eye and 6/12 in the left eye. She attended there for four months, but unfortunately there is no description of the lesion nor was a diagnosis recorded, but by the

treatment it was evidently thought to be an affection of the conjunctiva.

After that she attended a herbalist till December, 1925.

She then had a transverse growth across the left cornea six millimetres wide extending from limbus to limbus; the conjunctiva appeared free from any growth and the cornea above and below the growth was quite clear and transparent.

The growth itself was fleshy in appearance and raised from the cornea and had large blood vessels extending across it. Its appearance was like an overgrowth of conjunctiva across the cornea. The eye itself was congested and there was no palpable enlargement of any lymphatic glands.

She stated that the growth had been present for about two years commencing on the inner side of the eye and that in the last two months had grown considerably. There had been some pain earlier, but not for some time.

Operative Treatment.

The tumour was considered malignant and enucleation was advised as it would be impossible to preserve the sight. This was done on January 8, 1926.

At the operation a considerable portion of the conjunctiva on each lateral side of the cornea was removed with the globe. The conjunctiva was quite free and appeared normal.

One month ofter the operation she had one deep X ray treatment.

Pathological Report.

For the pathological report I am indebted to Mr. Henry Searby.

The *substantia propria* is normal in appearance. Bowman's membrane contains much well developed fibrous tissue. It is infiltrated by small round cells and all stages between these cells and formed fibrous tissue are present.

Opposite the most prominent epithelial proliferation the basement membrane between the epithelial layer and Bowman's membrane is lost, the basal layer of columnar cells has disappeared, but there is practically no invasion of the subepithelial tissues by the epithelial cells.

The *substantia propria* seems to form a very effective barrier to the downgrowth of the epithelium. It is in this region, however, that the round-celled infiltration of the *substantia propria* is most evident.

The proliferated epithelium is of the typical pickle celled type found in squamous carcinoma and cells are well marked. In this epithelial mass are spaces lined by endothelial cells and in some instances containing red blood cells. Round these blood vessels the epithelial cells are more columnar in type and grouped as they are on the basement membrane. Such an arrangement appears to be a nutritional effect, for the columnar type of cell changes a short distance away from the blood vessels into the ordinary epithelial cell of the growth.

Though a squamous-celled carcinoma of the conjunctiva and limbus is not rare and many cases have been reported, yet in the search through the literature of the last ten years at my disposal, I have been unable to see a similar case reported.

The usual method of extension of squamous-celled carcinoma of the limbus is along the sclerotic and often has a small attachment to the globe and forms a fungating mass.

The second point of interest is that although the tumour has been present for two years, the *substantia propria* has not been involved, showing the reason of the low malignancy of these growths.

Le Grange regards implication of the lymphatic glands as rare but it does occur and has been recorded. The treatment of these tumours depends to a considerable extent on their site. Treacher Collins[1] reports a case in which the tumour disappeared after one application of radium. Heckel[2] reports results of treatment by X ray therapy. Reese and Dixon[3] report cases treated with radium without success, but after the tumour was ligated, thus cutting off the blood supply, radium was reapplied successfully.

These methods of treatment are of use when the tumour affects the conjunctiva and a good eye may be saved, but when the growth affects the cornea and subsequent useful vision will not be saved then enucleation seems the better treatment.

References.

[1] E. T. Collins: "An Epibulbar Epithelioma which Completely Disappeared after One Application of Radium Bromide," *Transactions of the Ophthalmological Society of the United Kingdom*, 1915, Volume XXXV, page 215.

[2] E. B. Heckel: "Nonsurgical Treatment of Malignant Epibulbar Neoplasms," *Transactions of the American Academy of Ophthalmology*, 1921, Volume XXVI, page 149.

[3] Dixon and Reese: *Proceedings of the Section of Ophthalmology of the New York Academy of Medicine, American Archives of Ophthalmology*, 1916, page 279.

DR. H. F. SHORNEY (Adelaide) spoke on the treatment of carcinoma of the conjunctiva. He said that the coats of the eyeball were very resistant structures and that there was a temptation to try different methods of treatment, such as radium, before deciding to enucleate the eye. He had seen one patient in whom the conjunctiva was invaded by a small pimply growth; he had been tempted to advise excision of the growth, but it had afterwards been treated with radium. After some time as the other eye had started to water, he had enucleated the affected eye and had removed a wide area of conjunctiva. The growth had recurred and he had been compelled to remove the whole socket and the upper and lower lids in the end. He advised enucleation in all these cases.

DR. L. S. TALBOT (Timaru) had seen one patient who had had poor vision in the unaffected eye; in the good eye there had been a small growth close to the limbus with a history of three months' duration. The vision in this eye had been normal. He suspected it to be a malignant growth, had a piece excised and examined and it was found to be malignant. He recommended and did enucleation of the eye. He was glad to hear Dr. Shorney's opinion of the treatment.

DR. J. G. MACDONALD (Invercargill) was glad to hear Dr. Shorney's experience as to what ought to be done in these cases.

DR. A. G. TALBOT (Auckland) spoke on similar lines.

DR. W. E. CARSWELL (Dunedin) had had one case in a man of fifty years of age situated at the limbus like a small pterygium a millimetre in diameter and extending slightly on to the cornea. He had excised the growth and had taken care to remove a large area of conjunctiva; no recurrence of the growth had taken place since the operation nearly two years before.

DR. A. M. MORGAN (Adelaide) described a similar growth which he had excised, but it had recurred and he had been forced to enucleate the eye.

DR. A. J. HALL (Dunedin) described a case of a fleshy tumour mass extending from the conjunctiva on to the cornea six or seven millimetres in diameter. He had thought that it was malignant and had excised it. It had come away very easily, but on examination by Professor Drennan it had turned out to be a papilloma. There had been no recurrence.

SECTION IX.—PÆDIATRICS.

BLOOD TRANSFUSION IN YOUNG CHILDREN.

By EDGAR H. M. STEPHEN, M.B., Ch.M. (Sydney),
Honorary Physician, Royal Alexandra Hospital for Children, Sydney.

I HAVE compiled the results of blood transfusion obtained in a small series of patients at the Royal Alexandra Hospital for Children, Sydney, who were under my care. I have endeavoured to omit as much detail as possible in the belief that details are wearisome and, as a rule, unnecessary.

While I was in charge of the wards reserved for gastro-enteritis patients, intraperitoneal transfusions of citrated blood were given to eleven patients. Four of these recovered. As all were suffering from severe infections, I consider that this remedy deserves consideration. Three marasmic infants who had survived the ravages of gastro-enteritis, but whose low condition and anorexia still caused anxiety, made a rapid recovery in a few days after intraperitoneal transfusion. In two children in a very toxic condition wonderful results were obtained after intravenous transfusion.

1. A boy, aged four years, whose temperature was very high for nearly three weeks, following operation for double mastoid infection, within forty-eight hours of transfusion had a normal temperature and made an uninterrupted recovery.
2. A girl, aged three years, with acute exfoliative dermatitis, was in a desperate condition when an exsanguination transfusion was performed. Two hundred and eighty cubic centimetres were withdrawn and four hundred cubic centimetres were instilled. Except for a pyelitis which was present at the time, the patient made steady progress to recovery.

The course of the illness of eight children with chorea or acute rheumatism was very favourably influenced by transfusion. Exsanguination transfusion was performed on one, in the others no exsanguination was employed.

Six children with acute rheumatism and persistent anæmia were transfused. Some had an irregular temperature chart and all showed a very definite improvement which persisted. Enucleation of tonsils was then performed and the patients discharged in good condition, with a pulse normal in character and their anæmia a thing of the past.

Two patients suffering from chorea of severe degree were transfused.

The first had repeated exacerbations of rheumatism and pyrexia and heart involvement. After transfusion and tonsillectomy this debilitated invalid was discharged round and rosy and she has not returned.

The second with a paretic upper limb made haste to recovery after an exsanguination transfusion.

An infant of five months with idiopathic purpura was twice transfused. The signs were extensive, subcutaneous hæmorrhages in the abdominal wall and upper arms and extreme pallor. Over one hundred cubic centimetres of citrated blood were given through the superior longitudinal sinus and repeated in three days. No further hæmorrhages occurred. Blood platelets were definitely increased; the coagulation time was seven minutes at room temperature.

One of these patients suffered considerable distress after a transfusion of three hundred and forty cubic centimetres of citrated blood, the child being a normally developed girl of eleven years. I think two hundred and thirty cubic centimetres should have been the maximum utilized, as no exsanguination was employed.

A child, aged two and a half years, of Greek origin, with profound anæmia (red blood corpuscles 1,000,000 per cubic millimetre) became convulsed and died the next day. Its condition was so bad that one could not be surprised. Transfusions of the forlorn hope type to a protracted meningococcal meningitis and a pyæmic infant of six months, did not prolong life.

A reappearance of blood cells in the urine of an anæmic convalescent nephritic was of short duration.

DR. A. JEFFERIS TURNER (Brisbane) said that he hoped that dermatitis and acute rheumatism would not always be treated by blood transfusion. He had seen cases of hæmorrhages from the bowel of new-born infants cured by subcutaneous injection of blood taken from the parents. Apart from this his experience of blood transfusion was small.

DR. H. L. STOKES (Melbourne) had seen striking cures from blood transfusion. The method usually used was that of citrated blood, but in some schools they had obtained better results from the use of pure whole blood. He quoted experiments of Bruce Robertson who had introduced the method of exsanguination. The blood was taken from the child very slowly till the child became partially exsanguinated and the pulse weakened, then the new blood was given very slowly. At the end of the operation it was endeavoured to give a little more blood than had been taken away. He had seen good results from toxæmia of severe burns in erysipelas, septicæmia and in poisoning.

DR. F. M. SPENCER (Hamilton) mentioned a group of cases of pyæmic staphylococcal infection in which he obtained no good results.

DR. J. H. GRAHAM ROBERTSON (Napier) in referring to the case of exfoliative dermatitis mentioned by Dr. Stephen wished to record the fact that he had obtained good results in chronic skin diseases from blood transfusion.

DR. E. H. WILLIAMS (Dunedin) in referring to the intraperitoneal method of giving whole blood, quoted experiments done on animals in which blood was recovered from the thoracic duct twenty minutes after it had been injected into the abdomen. This showed that the absorption was rapid. It was not necessary to type the blood particularly when treating hæmorrhage of the new-born.

DR. G. H. USSHER (Timaru) said that in acute arthritis he had tried intravenous injection of saline solution, "Eusol" and "Mercurochrome" with no effect. He had used citrated blood with good results. In a case of ruptured ectopic gestation he had taken the blood from the abdomen and injected it into the median basilic vein with good results.

Dr. Stephen in reply thought that Dr. Jefferis Turner would use blood transfusions in acute rheumatism before long. He was of the opinion that all patients suffering from acute rheumatism did not require this treatment, but there were some who were treated with salicylates without avail and responded well to transfusion. There was no risk in doing transfusions provided the donors were typed. He did not think that transfusion required much manual dexterity, but it certainly took a good deal of time.

He stated that he had had no bad results. Occasionally a rise of temperature had been observed, but no rigors. In young children he had found a light general anæsthetic advisable, but with the help of the nurse and local anæsthesia children of five years and upwards could usually be managed.

In reply to a question regarding technique he stated that he always used gravity. The apparatus was well coated with paraffin.

SECTION X.—NAVAL AND MILITARY MEDICINE AND SURGERY.

DAYS AT AN ADVANCED DRESSING STATION.

By KENNETH F. GORDON, M.C., M.R.C.S. (England), L.R.C.P. (London),
Major, New Zealand Medical Corps; Ellerslie, Auckland.

IN presenting a paper of the nature I am now unfolding before you, I wish to state that it is very gratifying to me to have such an audience of veterans and also that so many of those present have been among my comrades in the Great War. I am especially pleased to see also a number of Australian medical officers here for, without minimizing the work of other units, I hold firmly to the belief that apart from their offensive work three times at least the situation on the Western Front was saved by colonial troops, namely by the Canadians at the second battle of Ypres, by the New Zealanders at Mailly-Maillet and by the Australians at Villers Bretonneux. The very nature of my audience prohibits the adoption of a lecturing style and if I adopt the narrative form of address, I am hoping to make myself clear and possibly to ward off criticism.

I want to say at the outset also how much I am indebted for material to Colonel Stewart's "Official History of the New Zealand Division" and Lieutenant-Colonel Carbery's "History of the New Zealand Medical Service" and to certain notes on field ambulance duties by Lieutenant-Colonel Hardie-Neil and if you should detect any happy descriptive phraseology during my remarks, you may justly surmise that I have purloined certain passages from these works.

My only excuse for addressing you at all lies in the fact that I did have a fairly varied experience in camp and field during the war, including two years in France. This must make some amends for the lack of a retinue of decorative lettering behind my name.

My military career, such as it was, began in this city some twenty-six years ago when I served as a private in the Dunedin Highland Rifles and therefore I have a peculiar pride in speaking here today. In France I acted as Regimental Medical Officer to the Second Canterbury Battalion for nine months and was attached to Numbers 1 and 2 Field Ambulance for varying periods. I was acting second in command of Number 1 during the period under review.

I do not propose entering very fully into the functions of an advanced dressing station. A field ambulance is a very elastic organization and in the work of an advanced dressing station the transport and quartermaster's departments are at least as important as what one may call the medical department.

The essentials of an advanced dressing station are (i) maintenance of communication with regimental aid posts, (ii) rapid collection and evacuation of casualties, (iii) maintenance of adequate supplies, (iv) efficient attention to the wounded.

The key-note of an advanced dressing station must, however, be evacuation and it will be seen later how bearer relay posts were established and cars were sent as far forward as possible to facilitate this work. The pooling of the ambulance transport and stretcher bearers of a division is an admirable conception to secure advanced dressing stations and regimental aid posts against congestion.

The duties of the quartermaster are particularly onerous during active operations owing to rations and supplies being required urgently in all directions. Requisitions come in from scattered regimental medical officers for blankets, splints, dressings and medical comforts and on the speedy fulfilment of their demands depends the expeditious and comfortable removal of casualties from the forward areas.

The wounded should receive such attention as will enable them to be carried on to the main dressing station in safety and comfort.

Particular attention must be paid to (*a*) hæmostasis; pad and bandage may be sufficient with packing if necessary; tourniquets may have to be eased and reapplied; car orderly must be informed of such cases; (*b*) application of splints; if already applied they may require readjustment; Thomas's splints must be used for all fractures of thigh; (*c*) administration of warm stimulants, such as well sweetened tea, cocoa, "Bovril" and the like; (*d*) administration of morphine where necessary; (*e*) application of warmth, by blankets, dry socks, hot water bottles.

I cannot overemphasize the value of training the tent subdivision of field ambulances. We certainly had a wonderful team of dressers in Number 1 Field Ambulance, so that supervision only by a medical officer was necessary for all ordinary work. Twice in particular I have had occasion to compare work of New Zealand dressing staff with that of other British units, much to our advantage.

Some months ago I selected the title of "A Day at an Advanced Dressing Station" for this paper. It would be more correct to call it "Days at an Advanced Dressing Station" but I shall enlarge more particularly on one day's experience, namely October 1, 1918, on which day the New Zealand Division in France captured Crêvecœur. On the same fateful day New Zealand machine gunners entered Damascus in Palestine, while at Spa, in the Ardennes, Ludendorff was solemnly announcing to his War Lord that the end had come and that Germany must sue for peace.

For the purposes of this paper I am exhibiting three maps, two being official war maps of the area under consideration and the third a rough enlargement of the particular field of operations, the scale

being approximately 500 yards to the inch. On this I propose to indicate the positions of the advanced dressing station, bearer relay posts and regimental aid posts.

In default of depicting the contour of the terrain I must explain that the area between Trescault and the Canal de l'Escaut consists of broken country, a succession of ridges and valleys, the ridges reaching a height of 150 to 200 feet. If one can imagine the area to the south of Gouzeaucourt as a plateau represented say by the wrist, then the fingers outstretched and prolonged to the north might represent in succession the Trescault, Beaucamp, Highland, Welsh and Bonavis ridges. This area is roughly bisected by the Couillet Valley. The distance between Trescault and the canal is roughly six miles. The canal itself is nowhere fordable. On the far or eastern side of it the ground rises steeply behind Crêvecœur to the La Terrière plateau which is broken by sunken roads and quarries and from the plateau a broad spur overhangs Vaucelles. Cheneaux Wood climbs up the slope.

Rugged as the country is, its negotiation was rendered more difficult by the maze of trenches and wire entanglements which constituted part of the Hindenburg system.

It was this tract of country embracing the two canals (du Nord and de l'Escaut) fortified by nature and by art and the high sounding Siegfried and Wotan lines, which were referred to in the German Reichstag in September as "the wall in the west which would never be broken." A week later No. 1 New Zealand Field Ambulance established an advanced dressing station in substantial dug-outs in the centre of this Hindenburg line and New Zealand Infantry had passed through the impenetrable wall. A feature of this map to which I will direct your attention, is the trend of the roads northward and southward, a feature which, especially on the New Zealand Division's area did not facilitate evacuation and necessitated long carries by the bearers in the earlier days under review.

It is essential I think, that a very brief indication should be given of the scheme of operations during the days in question, namely, September 28 to October 8.

The First and Third Armies were advancing in the general direction of Cambrai, preparatory to a grand attack by the Fourth Army on the right (or south) of them. The objective of these three Northern Armies was the seizure of that knot of railway communications which is included in the triangle Valenciennes—Maubeuge—Mons. This was the central ganglion of the German communications that controlled the maintenance of their armies in France from Lille to Metz. The New Zealand Division, together with the Forty-second, Fifth and Thirty-seventh Divisions (British) constituted the Fourth Corps of the Third Army. The Sixth Corps and the Seventeenth Corps (Third Army) were attacking on our left (or northern) flank. The next Division to ours was the Sixty-second, later succeeded by the Third. The orders of the Assistant Director of Medical Services issued on September 28 were so precise and yet so comprehensive, that I shall presently quote them in full, but for the information of our Australian guests I would like to state that our First Brigade consisted of two Auckland and two Wellington Battalions, our Second Brigade of two Canterbury and two Otago Battalions and our Third (or Rifle) Brigade, though equally distinguished, did not retain any provincial characteristics.

In the beginning of the advance the Second Brigade was on the right and the First on the left, while the Rifle Brigade was in reserve. Later the Rifle Brigade relieved the First Brigade and carried on the advance to the La Targette-Esnes line, the Second Brigade still cooperating on the right.

These are the orders to which I have just referred.

[COPY.]

SECRET. COPY No. 11.

NEW ZEALAND DIVISION.

Headquarters, 28th Sept., 1918.

N.Z.M.C. Order No. 71.

by

Colonel D. J. McGavin, D.S.O., A.D.M.S., New Zealand Division.

Reference Map:—57.D: 1:40,000.

1. Information:

(A) On the night September 28th/29th the New Zealand Division will pass through the 42nd Division and continue the advance.

(*a*) To complete the capture of the Bonavis Ridge;

(*b*) To secure the bridgeheads across the Canal de St. Quentin and Escault River between Vaucelles and Crêvecœur sur l'Escault, both inclusive.

(B) In the event of La Vacquerie and the Bonavis Ridge being in the enemy's possession, the 2nd New Zealand Infantry Brigade (on right) and 1st New Zealand Infantry Brigade (on left) will attack in conjunction with the 5th Division on that night by moonlight. Zero hour will be 3.30 a.m.

(C) The 3rd Rifle Brigade Group will be prepared to move at 6 a.m. on September 29th to relieve reserve brigades of 42nd Division in an area east of Trescault.

2. O.C. No. 1 New Zealand Field Ambulance will:

(*a*) Establish advanced dressing station at Trescault forthwith;

(*b*) Detail one bearer squad and runner to each battalion of 1st and 2nd New Zealand Infantry Brigades forthwith;

(*c*) Place bearer relay posts and car posts at suitable locations with reference to regimental aid posts when these are established.

3. O.C. No. 2 New Zealand Field Ambulance will take over the main dressing station at Ruyalcourt from 42nd Division forthwith.

4. Evacuations of wounded from R.A.P.'s to M.D.S. will be by hand carriage, wheeled stretchers and field-ambulance cars. O.C. No. 1 New Zealand Field Ambulance will have at his disposal all cars of No. 2 New Zealand Field Ambulance and all except two large cars of No. 3 New Zealand Field Ambulance. These cars will be sent forward to Trescault by O.s C. Nos. 2 and 3 New Zealand Field Ambulances forthwith. He will also have at his disposal the horsed ambulance waggons of Nos. 2 and 3 New Zealand Field Ambulances.

5. From the Main Dressing Station stretcher cases will be evacuated by cars of 21st M.A.C. and walking cases by:

(*a*) Light railway from walking wounded entraining post at Ruyalcourt (P.9.d.8.2.);

(*b*) By buses and lorries provided by 21st M.A.C.

6. The A.D.S. will be established and be prepared to open at 5 a.m. on 29th September.

7. Office of A.D.M.S. will open at Velu on 29th September at an hour to be notified later.

8. New Zealand Field Ambulances to Acknowledge.

(Sgd.) D. McGAVIN, Colonel, A.D.M.S.
New Zealand Division.

On September 29, therefore, our advanced dressing station was opened at Trescault at 5 a.m. and runners and bearers were dispatched to the First and Second Brigades. Cars were pushed forward where possible and bearer relay posts established. On the map I have indicated for this day the position of the advanced dressing station and regimental aid posts only.

At 6 p.m. we had evacuated 308 wounded, including Germans. Shells had fallen in our vicinity during the day and at 7 p.m. owing to persistent shelling we were ordered to establish advanced dressing stations towards Ribecourt. Here capacious dugouts afforded cover and shelter. Before midnight another hundred wounded had been evacuated. Incidentally the New Zealand Division had captured 1,400 prisoners, two naval guns, thirty field guns and over two hundred machine guns, and of these Second Otago had taken nearly 600 prisoners with thirty officers, four field guns, two trench mortars and thirty machine guns. Two other features of the morning's work may well be recalled.

The rapidity of the advance of the First Brigade. Zero hour was 3.30 a.m. and by 6 a.m. the two forward battalions had crossed the Cambrai road in the direction of the canal.

The prospect enjoyed from Bonavis Ridge whence the grey lofty towers and spires of Cambrai were clearly visible and great beacons of smoke told of the destruction of dumps or the firing of the city. Behind the troops now was the lacerated familiar landscape of churned up shell-holes, hideous walls of wire, miry battered ditches, the unsightliness of stricken villages and blasted woods.

Eastward stretched a smiling country unscathed by war. "The attack had reached the threshold of a new world," writes Colonel Stewart, "and fatigue was forgotten in tense exhilaration."

During this day frequent demands came in for stretchers, dressings and splints, while extra bearers were sought by the Second Auckland. Time forbids further details until October 1, the day of the crossing, approached. Owing to the energy and ardour of my Commanding Officer, Lieutenant-Colonel George Craig who, to quote R. L. Stevenson "courted danger like a mistress," I was denied the opportunity of doing forward work at this time. Hence I received at the advanced dressing station over thirty communications during the day from regimental medical officers and forward officers and others and I propose submitting some of these to you as an indication of the work of an advanced dressing station. Also if time permits, I shall offer some brief comment thereon. The new positions of regimental aid posts, bearer relay posts and car posts I now indicate on the map. My rough diary notes of the day's doings reads as follows:

October 1, at K 36, *c.* 22.

Weather, fine but keen.

C.O. forward at car post and a new one established south of Masnières.

Capt. Baxter moved from 1st to 2nd car post and transmitted six-hourly reports.

At 11.30 a.m. runner from 1st A.I.R. informed me of twelve stretcher case casualties. A.D.M.S. reached A.D.S. at the same moment and told me he had not been informed of battle operations in due time. There was five hours' delay at signals. Continued to receive scare messages from 1st Brigade and it was reported to me that there were 60 cases at 1st A.I.R., R.A.P.

A.D.M.S. and Colonel Avery, A.A.Q.M.G., informed me that 50 O/R from 3rd Brigade were detailed to a point and I forwarded stretchers, blankets and guide (Voss).

Later was informed no such infantry appeared.

Continuously busy at A.D.S. from noon till 5 a.m. on October 2. The greatest number of stretcher cases at one time was 32. O.C. returned about 6 p.m. for ten minutes and met A.D.M.S. and proceeded to reconnoitre Masnières vicinity.

Returned about 11 p.m.

Acting on instructions from A.D.M.S. I had sent Lieutenant Spencer forward about 5.30 p.m. to Captain Baxter, thence to help Captain Ardagh (1st A.I.R.) and report. He returned with favourable report about 9 p.m.

Capts. Dempster, Simmonds, Anderson and Lt. Spencer very busy all day dressing cases.

Old car post was quite clear at 7.30 p.m. Cars to and from new car post continuously through night.

Received communications from R.M.O.'s as to locations supplies *et cetera* during day and attended to same.

Capt. Baxter's communications very acceptable as to forward locations.

Pioneer party returned to A.D.S. after acting as bearers at B.R.P. and 1st C.I.R.

Warned during the night that we were to open A.D.S. at car post site at 10 a.m.

October 2, breakfast for all hands at 6.30. C.O. took forward party to the new site (G 25 c. 82) and self instructed to remain until personnel forward. A.D.M.S. visited new A.D.S. about noon.

Some shelling round about A.D.S. in the evening.

Capt. Wyllie rejoined unit during night.

Copies of some of the notes delivered by runners to the Advanced Dressing Station; operations on October 1, 1918.

Written at 11.30 p.m. on September 30, 1918.

1st Auckland R.A.P. moving forthwith to approximately G 27 central east of Masnières—exact reference will be supplied later. Could B.R.P. be brought forward to present R.A.P. location that is at M 2 a 47, otherwise bearers will not be able to cope with evacuation as a busy time is expected after 6 a.m. tomorrow.

ARDAGH,
Capt. R.M.O., 1 A.I.R.

Have two stretcher cases here and location makes it impossible to carry by night. Could you send car to collect cases.

MULLHOLLAND,
1 a.m. Capt. 1st C.I.R.

R.A.P.—R 22 c 97.

Send car or wheel stretchers to R 14 a 08. Four urgent cases.
1 a.m. R.M.O. 1st O.I.R.
3 a.m.—R 18 a 68.

I am moving forward first thing in the morning at dawn and will let you know later my location. Should strongly advise you to get cars forward as the carry is already a very long one, at least from four or five miles for your bearers. Cars can get here by going up north to Marcoing and then south down the valley. The bearers (8) living here received no rations today,

though I saw Colonel C. about it yesterday morning and he promised to get them rations. They went down for rations to the car post, I believe. I managed to scrape up a meal for them last night. This place should be made a B.R.P. when I go forward and I shall leave the bearers to occupy it.

REID,
R.M.O., 2nd W.I.R.

Regimental Aid Post at G 27 central.

Please send stretchers, bearers and dressings urgently, as there are already twelve cases waiting here and many more are expected.

ARDAGH,
8 a.m. 1st A.I.R.

Urgent.

Please send immediately to R 3 a 42 three heavy cars and two Ford cars to clear wounded. This is position of new car post: also three horse ambulances for walking cases will be sent here immediately. The route is through Ribecourt to Marcoing and thence to the right along the Gouzeaucourt Road. This is a good road.

G. CRAIG,
11.10 a.m. Lt.-Col.

Instructions carried out at 11.25—

K. F. G.

3/2463 Pte. McAllister G. reports to you herewith. He fell into a shell hole last night and cut his knee. I should be glad if you could replace him forthwith.

Would you please advise me if you have any word of moving your B.R.P.S. and car posts.

GIBSON,
R.M.O.,
1st O.I.R.
R 8 d 84.

We are shifting our R.A.P. up to 1st Auckland Hd.-quarters as their M.O. is missing. Map reference as for us I know is Masnières. Plaisir Farm.

We are in need of dressings.

HASKINS,
1st W.I.R.

I am sending on my 2nd squad of bearers to 2nd Canterbury R.A.P. at R 18 a 60. The 2nd C.I.R. are taking over the front line work from this Battalion.

I am keeping one squad and a runner with me and these are on our ration strength. The squad I am sending to 2nd C.I.R. will require to be rationed from your A.D.S.

R.A.P. is still at R 3 d 30.

GORDON,
R.M.O.,
2nd O.I.R.

Location 2nd C.I.R., R.A.P. as before R 18 a 52.

I beg to report that four bearers transferred from 2nd O.I.R. (Pvts. Norton, Pollock, McKenzie and Parsons) have reported to me for duty.

MARKS,
2nd C.I.R.

To Major Gordon, A.D.S.

I have just returned from a tour. Saw Colonel at Car Post. He has gone up to look at a new car post.

I have suggested G32—C64. This is at present strategical point in the evacuation of whole sector. It is right beside 2nd A.I.R. R.A.P.—1st A.I.B. is in G 27 b 2nd W.I.R. I have not seen, but think they are in M 2 b. The right Brigade R.A.P.'s can evacuate easily to G 32 c along paved road from Bois Lateau, M 14 a by means of wheeled stretcher. I have the Colonel's permission to ask you to send a car with rations to Sergt. Perry at G 32 c 64. This is urgent. Rations are short all round, our bearers (over and above original squad of 4 men) with 1st and 2nd A.I.B. have had no rations for two days. This car could go through Marcoing as the road between Marcoing and G 32 c is excellent. Most of R.M.O.'s are crying out for blankets, splints and medical comforts. Could you establish a dump of stretchers and blankets with me at G 32 c.

Harpur has nine stretcher cases. Ardagh has twenty stretcher cases awaiting evacuation. There are still some to come in.

I enclose indent from 2nd A.I.R. which is urgent. Most urgent rations and water for forward posts. Send plenty because we have to give some to our Hun bearers.

I'll double your three, no trumps.

1.40 p.m. BAXTER,
Capt. N.Z.M.C.

Just received your memo. re Sergt. Sclater and party. I would like to keep the Pioneers until we get R.A.P.'s clear.

1st B.R.P. now at G 32 c. Sergt. Perry.

2nd B.R.P. now at R 10 b. Sergt. Hickey.

Cars are now evacuating from Hickey's old post R 3 c 19. I am now going back again to G 32 c. Personnel of car post will move up to R 3 c 19 as soon as we are clear here. There are at present fifteen sitting cases here at old car post R 2 b 19. I shall let you have six-hourly reports.

2.15 p.m. BAXTER,
Capt. N.Z.M.C.

To Major Gordon,

Please send urgently all available cars to new car post R 3 a 23.

2.30 p.m. CRAIG.

We are moving in about three-quarters of an hour's time. Could you send bearers?

2.45 p.m. HORSBURGH,
3rd N.Z.R.B.

As you no doubt know, things did not work out exactly to plan. There was a big congestion at 1st Auckland R.A.P., so I took my bearers up there. The bearers who were at that post were done up, so my men have relieved them. Things were a lot better when I left that post about 5.30.

SCLATER, S/Sgt.

More bearers required with 2nd Wellington and 1st Auckland. Sixteen additional squads if possible; insure that there are always two cars with advanced car post G 32 b. 05.

4.45 HOLDERNESS,
Staff Captain, 1st Brigade.

A party of 50 infantry will rendezvous at G 26 c 11 road junction A A A. These are for purpose of stretcher bearing A A A. Arrange supply of stretchers and guides for this party.

Time ? D. MCGAVIN,
A.D.M.S.

The two squads and runner (nine men in all) working with 2nd Wellington I.R. have within the last 48 hours received only one bag rations for four men. Send up plenty of rations tonight certain with car as men are working hard on no food. Mark bag 2nd W.I.R. nine men.

L/c STAPLEY.

My R.A.P. now is 57 C R 3 c 76.
(In reserve)

PATERSON,
1st N.Z.R.B.

Received fresh party of four bearers to replace Pioneers at 53.0 p.m.

The four Pioneers will report as they leave here for your A.D.S..

5.45 MULHOLLAND,
R 22 b 97.

The fifth Pioneer who, without my knowledge or consent went out as I learn, to get water from some well near by, evidently leaving soon after noon, and up to present has not returned and I can get no information regarding same.

5.45 MULHOLLAND,
1st C.I.R.

Bearers on ambulance ration strength have not received any rations. Can this be remedied please?

ARDAGH,
1st A.I.R.

Somebody has the wind up. Capt. Ardagh is not missing. Both he and Capt. Reid are in G 27 b. All cases are clear of these aid posts except a few Huns.

The right Brigade sector is clear. I shall keep Spencer to supervise evacuation of right sector.

Car post is working beautifully.

6 p.m. BAXTER, Capt.

Everything is set. Last report from 1st A.I.R. was one stretcher case at R.A.P. I have seen Staff Capt. 1st Brigade and he says the wounded are out in front of R.A.P. not at R.A.P.. This is how the scare commenced and, of course, it is not our concern. Have refused to send my bearers in front of R.A.P.; but told him I would give him every assistance with stretchers and blankets. I am now going up to establish a B.R.P. for right sector near Bois Lateau.

7.10 p.m.

Urgently required:

(i) Stretchers, (ii) blankets, (iii) two red lights, (iv) water. Car waiting post G 25 central. Car loading post G 32 a 92. B.R.P. and main H.Q. G 32 a 64.

Car and stretcher directing post (two men) at corner G 32 b 05. Driver Hill has arrived with his load of rations.

I say with perfect confidence that there is no congestion and never has been any marked congestion between R.A.P.'s and A.D.S. Tomorrow morning I intend to relieve some tired bearers by the fresh ones. The tired ones I shall send back to A.D.S. Can you let me know 3rd Brigade R.A.P. locations? I have instructed R.M.O.'s of 2nd and 3rd Battalions, but have not seen R.M.O.'s of 1st and 4th.

Harper 2nd A.I.R. has moved forward, but I have not yet received his location.

1/10/18, 8.50 p.m. BAXTER, Capt.

8.50 p.m.

We have fifteen stretcher cases and fifteen sitting cases at car loading post. Please do not delay cars. We expect cases in during the night. This is urgent; we can use four big cars through the night.

1/10/18. BAXTER, Capt.

O.C., A.D.S.

I have personally visited all R.A.P.'s of 1st Brigade this morning.

1st A.I.R. G 27 b central. One case being dressed. Bearers waiting to take it down. Corporal and two squads attached from ambulance.

2nd A.I.R. G 33 a 10. No cases during the night, no cases waiting. One squad attached.

1st W.I.R. G 32 b 11. No cases during night, no cases waiting evacuation. One squad attached.

2nd W.I.R. G 27 b central. No cases awaiting evacuation. One squad attached.

Squads indicated above are over and above permanent runner and squad.

I have established a B.R.P. at Canal just across Lock (G27, c 37) Corporal and three squads.

Car loading post has been moved slightly to G 32 b 11.

This place is an excellent site for a future A.D.S.

Sergt. Cardno is reconnoitring. 2nd Brigade R.A.P.'s

Will you please inform 3rd Brigade H.Q. of my locations? Only one 3rd Brigade R.M.O. is at present in touch with me.

2/10/18. 11.30 a.m. BAXTER, Capt.

1. Please look at Pvtes, Warren and Dennis, N.Z.M.C., who the Corporal states are not able to do full duty on account of bad feet and being played out.

2. Re Socks. Some of bearers have blistered feet *et cetera*, which is probably due to having no change of socks. They say they have only those they are wearing. Until they can change socks each day, I am afraid some of them will crock up.

3. Things quiet here and O.K. Tomorrow may be busy.

ARDAGH,
Capt. 1st A.I.R.

The following bearers report back to A.D.S. for a rest: Lees, Moore, Langford, Brighting. Paine has a bad foot and has been ordered back by Capt. Baxter. Voss will be sent down later on.

Things are clear now and 1st Auckland has only three cases. Dooley has a slight wound on the hand and was sent down by a previous car for A.T.S. He also needs a rest.

J. SCLATER
S/Sgt.

O.C., A.D.S.

Would it be possible to have one of the Company cooks up here to cook for the men at the car post and the reserve bearere? At present we have a man (not a cook) cooking for the whole crowd, about 50 men. We would also be glad of three acetylene lamps and carbide.

If you could send up a water cart we would keep it under cover, but the horses would need to go back each day.

4 p.m.

Later.

Please if possible let me have one pack of cards for relaxation.

I have changed my headquarters to G 32 b 11. Car loading post.

BAXTER, Capt.

In confirmation of Captain Ardagh's note referring to the fatigued condition of some of the bearers, I recently met one of the men named in note No. 25. He recalled some of his experiences including a night in the remains of a roofless building on the outskirts of Masnières. Careless of orders the party of bearers were grouped around a lighted candle when two bombs were dropped from a German aeroplane in their immediate vicinity. The concussion from one knocked some of them over and from the other dislodged Captain Wylie from the seat of a car. The application of some welcome medical comfort, however, quickly restored the equilibrium of the Medical Corps. Another night he spent in Captain Ardagh's dugout and this officer let the exhausted bearers sleep for two hours before they cleared some enemy casualties. A third night he slept in the open regardless of the rain, absolutely worn out.

He stated that his party made good use of prisoners for the conveyance of wounded.

For the condition of his feet, he considered himself to blame for not carrying the regulation extra pair of socks with him. He never wanted for rations. He described afresh the broken nature of the country traversed, the wire entanglements and some especially wide and deep German trenches in the Hindenburg line, evidently designed for defence.

The principal comment I have to offer on these notes is the evidence they provide of close communication with regimental medical officers, of the requirements of these officers in moving warfare, of the activity and initiative of forward evacuating officer (and I have said all too little of the work of the non-commissioned officers similarly engaged) and of the cooperation of all ranks from Lieutenant Colonel to Lance Corporal in the service of the wounded. The solicitude of various officers for the condition of the men, their rations and their fatigue and so on is also very noticeable and this solicitude to my mind is an essential for army discipline.

The reports of Captain Baxter speak for themselves and to Colonel Craig's ceaseless activity and supervision the success of the Advanced Dressing Station on October 1 was principally due.

Criticism of Captain Baxter's refusal to send ambulance bearers in front of the Regimental Aid Post may be forthcoming, but he was technically correct, although he did not know that the strength of the First Auckland which approximated fourteen officers and 500 men on entering into action was reduced to five officers and 157 men on relief on October 3. It is satisfactory to note that on the morning of October 2 he reports only one patient at the Regimental Aid Post in question.

I cannot refrain from drawing attention to the notes from Captain Ardagh of the First Auckland. It is gratifying to know that for the fortitude he displayed in attending to wounded at an inadequately protected dressing station for thirty-six continuous hours under the heaviest shell fire he was recommended for a V.C. and granted the D.S.O.. I shall close my paper with final extracts from diary notes of October 8 and once more set out the positions during the closing stages of these operations.

DIARY, 8th October, 1918.

Wounded began to come in about 6 a.m. and there was a continuous stream until after midnight. Approximately 800 passed through the A.D.S., nearly 200 being Huns and over 200 being British.

O.C. was forward the greater part of the day.

A.D.M.S. visited A.D.S. twice during the day.

Voluntary assistance was given by M.O. attached to a Tank Battalion and also by Capt. Richards, N.Z. Tunnelling Co.

Positions were as follows:

A.D.S.	G 25 d. 82
Car Post	G 29 c. 30
Do.	M 11 c. 39
B.R.P.	N 1 c. 30
R.A.P.'s	
1 A.I.R.	R 5 a. central
2 A.I.R.	R 10 c. 91
1 W.I.R.	R 9 c. 54
2 W.I.R.	R 3 c. 52
1 C.I.R.	M 11 c. 76
2 C.I.R.	N 3 a. 95
1 O.I.R.	N 1 c. 30
2 O.I.R.	N 1 b. 12
1 N.Z.R.B.	G 36 b. 39
2 N.Z.R.B.	H 26 d. 57
3 N.Z.R.B.	(to G 30 a. 53)
4 N.Z.R.B.	G 33 c. 19

The Rifle Brigade and the 2nd Brigade are now in the lead on a narrowed front having secured their objectives and established posts in the direction of Beauvois and Fontaine where the Division rested from its labours.

During the period September 29 to October 12, 75 officers and 1,934 "other ranks" passed through the dressing station and the Assistant Director of Medical Services reported that the evacuation of wounded had invariably been carried out rapidly. This was attributed to the satisfactory condition of the roads, the pushing forward of car posts and the experience of the New Zealand Medical Corps personnel. Close touch with regimental aid posts and daily personal visits by Colonel Craig and Captain Baxter and later by Captain Wyllie were also factors securing smooth and rapid exacuation.

If one may add that medical supplies were maintained to meet all requisitions and that the wounded reached the main dressing station in satisfactory condition, one may conclude that the principal functions of an advanced dressing station were duly fulfilled.

Accounts of later operations, including the capture of Le Quesnoy, will be found in the works to which I have referred. That exploit is described with peculiar charm by Colonel Carbery and finally I quote him as an authority for the statement that, during the occupation the chief pleasure of the New Zealand Medical Corps in Cologne was at the Opera House where they heard in song the great Saga of our Norse ancestors, saw and heard Siegfried forging his sword and Brunhilde, daughter of Wotan, riding the winds with the choosers of the slain, saw again as at Crêvecœur and at La Vacquérie, the twilight of the Gods and the doom of Valhalla.

SECTION XII.—RADIOLOGY.

MALIGNANT DISEASE OF THE SKIN.

By P. CLENNELL FENWICK, C.M.G., M.D., F.R.C.S. (Edinburgh), F.R.G.S.

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CANCER of the skin is most conveniently classified into two varieties: basal-celled or rodent cancer and squamous-celled or prickle-celled cancer, usually known as epithelioma.

These two cancers differ so much both in microscopical appearance and in their life history that a careful study of their peculiarities opens a wide field of conjecture as to the origin of the malignant invasion.

TABLE I.—MICROSCOPICAL APPEARANCE.

Basal-celled Cancer.	Squamous-celled Cancer.
Solid columns of epithelial cells growing downward into the connective tissue.	Solid columns of epithelial cells growing downwards into the connective tissue. columns become thinned out and form prolongations
These columns reach the same level. They show no tendency to push themselves into the intercellular spaces of the tissue invaded.	The extremities of the which push themselves into the intercellular spaces of the invaded tissue.
No cell nests are seen.	Cell nests are prominent.
Keratinization is absent.	Marked keratinization present.
The surface of the ulcer is covered with a layer of epithelial cells.	The epithelium is pushed aside or destroyed by the growth. The surface of the ulcer is formed by tumour cells.
Cells are similar to the basal cells of the epithelium, are arranged in palisade formation, are small and compact.	Cells are enlarged, irregular in shape and are arranged in irregular formation. May be multinuclear.
Nuclei stain deeply.	Mitotic figures are irregular or multipolar.
Cytoplasm is scanty.	Cells are studded with droplets of Eleidin.
Nuclear irregularities are absent.	Nuclei stain deeply.

The life histories of these two varieties are sharply contrasted. Basal celled cancer is painless in its origin and throughout its history unless it becomes infected. It seldom becomes septic and even in its most advanced stage does not smell offensively. It shows no tendency to affect the neighbouring glands or to produce metastatic deposits.

Squamous-celled cancer is painless in its origin, but when ulceration commences, becomes painful. This form of ulcer rapidly becomes septic and smells most offensively. The growth infiltrates the neighbouring tissues and glands and metastasis occurs.

Site of Selection.

If a line is drawn from the angle of the mouth to the tip of the ear, it is stated that the great majority of growths above this line is of the basal-celled variety. Below this line the majority is of squamous-celled type. Rodent ulcer is not often found in other parts of the body. Out of two hundred cases seen in this department, one occurred on the buttock and two on the hand. The portion of the face above the line mentioned above is supplied by the fifth nerve and this, I think, is significant.

The fifth nerve has most intimate relations with the sympathetic nerve system.

The portio major, sensory root, passes into the Gasserian ganglion, which has been compared to the ganglion on the posterior root of a spinal nerve, which it probably resembles in exercising a trophic influence on the nerve fibres. The Gasserian ganglion receives a communication from the carotid plexus of the sympathetic. (Morris.)

It has been our routine practice for many years to test the sensation of the skin round all ulcers and almost invariably we find an area of about one millimetre in width which is insensitive to the prick of a fine needle. As soon as the ulcer responds to radium treatment, this insensitive margin becomes hypersensitive.

The growth of these two forms of cancer is also in sharp contrast. The rodent cancer extends slowly, with a tendency to remain superficial for a long time, the superficial fascia appearing to act as a barrier. Later the ulceration extends into the deeper portions of the tissue, causing horrible destruction.

The squamous epithelioma spreads much more rapidly. The destruction of the surrounding tissues is not limited to the superficial layers. A deep, foul-smelling irregular shaped ulcer forms and spreads with remarkable rapidity.

I have watched cases of rodent cancer during a period of years. I have under observation three patients in whom enormous destruction of the face has occurred, but a curious fact has been noted in all. The rodent cancer does not spread across the mid-line of the bridge of the nose. The ulcerated area stops exactly in the mid-line and no destruction of the opposite side of the face has occurred.

A second fact has been noted. I have records of many cases in which the ulcer has originated in a small pimple in the hollow between the eye and the nose. The direction of the ulceration always appears to be towards the inner canthus, as if the ulcer were "tracking" towards the eye, following the course of the infratrochlear nerve. In other cases in which the ulcer has begun above the eyebrow, the direction of the ulcer seemed to be downwards toward the supraorbital notch. When healed by radium, a racket-shaped scar is left, the handle of the racket pointing towards the inner canthus or the supraorbital notch respectively.

These facts which I have frequently noted, would appear to suggest that the trophic nerve supply to the skin has some intimate relation to the direction of the spread of the ulceration.

I have never seen sepsis in a true rodent cancer, but septic infection of a squamous cancer is common. I believe that this contrast is due to the fact that rodent cancers retain a covering of epithelial cells over the surface of the ulcer which may act as a protection against the entrance of bacteria. The absence of this epithelial cover in squamous cancer would render the ulcer open to bacterial attack.

I have constantly recorded the absence of presence of "smell" in my notes and find, I believe, invariably that rodent ulcers are quite free from offensive odour, the only smell being that like "dead meat." On the other hand, squamous-celled cancers frequently smell horribly.

I have ventured to say that I could diagnose the variety of skin cancer by the absence or presence of offensive odour in the ulcer.

I believe that this difference may be explained by the fact that the rodent ulcer is derived from the deeper layer of the epithelium and shows no keratinized cell nests, while squamous cancer originating in the horny layers, has cell nests rich in keratin.

Keratin abounds in sulphur more than any other protein and one would naturally expect that sulphuretted hydrogen would be produced by decomposition of this class of tissue.

For many years I have believed that the vulnerability of the tissues to cancerous invasion is dependent upon alteration or destruction of the trophic nerve supply to the invaded area.

If we grant that the cancer virus is universally present, like the spirochæte or tubercle bacillus, there must be some reason why certain patients are invaded and others are not.

I suggest that we may reasonably learn some lessons and suggest some theories from watching cancer of the skin. I suggest that the life history of skin cancer might be written on some such lines as follows.

An area of the skin, a sebaceous gland or a sweat gland is destroyed by chronic irritation, blockage by dirt or any cause. This area being functionless, becomes a useless factor in the body; in effect a foreign body. The trophic nerve supply to this area being no longer required for the maintenance of health in this useless area, atrophies.

I suggest that this atrophy of nerve fibrils would explain the area of anæsthesia round the rodent ulcer I have noted.

This dead area which I suggest is the point of attack by the cancer virus, is represented by our term, precancerous lesion, the dry patch of keratosis, the chronic wart or crack, the scar of a lupoid or syphilitic lesion or a portion of skin chronically irritated by tar or dyes. This condition may remain for years a latent danger which becomes potent when invasion by the virus occurs. Dr. Strangeways has recently given a lecture on the living tissue cell and tabulates the reactions of the cell to its environment, as follows:

1. Multiplication *plus* differentiation *plus* coordination *plus* inhibition = animal.
2. Migration *plus* inhibition = healing by first intention.
3. Migration *plus* multiplication *plus* inhibition = healing by second intention.
4. Multiplication *plus* differentiation *plus* inhibition = innocent tumour.
5. Multiplication *plus* differentiation *minus* coordination *minus* inhibition = cancer.

I suggest that the withdrawal of the nervous supply of the precancerous area fulfills the conditions of want of inhibition and loss of coordination mentioned by Dr. Strangeways.

The virus by its own reaction on the invaded tissue produces its required specific chemical factor. This factor should by biochemical reasoning be different according to the chemical constituents of the various cells and thus a cell containing keratin should produce a different specific factor from a cell which does not contain a similar constituent.

I suggest that this would explain the difference in the behaviour of a prickle cell from that of a basal cell cancer.

The effect of radium treatment upon the two classes of skin cancer is interesting and leads to certain queries.

It has now been accepted that the more undifferentiated or embryonal type of cell reacts much more easily to radium than the cell whose differentiation is more advanced. Thus the basal cell cancer is certainly more amenable to treatment than the squamous. Secreting cells are more sensitive than non-secreting and this is easily proved by the action of radium on an area well supplied by sweat glands. We have constantly found that the axillæ or palms of the hand react most strongly to a dose of radium that will scarcely cause reddening on the back of the hand or arm.

I suggest that the fact that rodent ulcer reacts so favourably to radium treatment, while twice the dose will be required to cause reaction in a squamous cell epithelioma might cause us to inquire if the rodent or basal cell cancer is not really a malignant disease of a secreting gland and that the suggestion that rodent cancers arise from the basal cells of the epithelium is not a mistake.

I offer as a suggestion that the disorderly downgrowth of the squamous cells are explained by the fact that these cells are in intimate contact through the prickle-like processes which join the cells in this layer, affording an easy channel for the spread of the virus, while the curious regular columns seen in rodent or basal cancer are due to the fact that the disease is limited by the basement membrane on which the basal cells rest.

If my suggestion that nerve atrophy precedes the invasion of the malignant virus is accepted, would not this explain the painless onset of cancer which has always been a puzzle to clinicians. The extreme pain which is seen in the squamous cell cancer is, I think, due to the septic infection causing nerve irritation far beyond the limits of the ulcer. I have often noted that this pain is relieved by radium and have attributed this relief to the bactericidal action of the rays.

Rodent cancer does not infect the neighbouring glands. Squamous cancer does.

I offer as a theory that this is due to the fact that the rodent ulcer, protected by the intact layer of epithelial cells spread over its surface, is safe from bacterial invasion. The epithelioma whose surface is sooned or later ruptured by the growth of the tumour cells, is invaded and becomes septic. The resulting inflammation produces *per se* increased vascularity with formation of more vascular channels and the malignant virus is carried far and wide.

I further suggest that the slow growth at the edges of the rodent ulcer seals the lymphatic channels, exactly as we seal these with the needle points when removing a growth by endothermy.

As a summary I offer the following suggestions:

1. That the living body cells are able to resist the invasion of the malignant virus as long as their trophic nerve supply is intact.

2. That the death of any portion of the body tissues is followed by the atrophy of the trophic nerve supply to that part. The dead portion then becomes a pre-cancerous lesion.

3. The withdrawal of the trophic nerve supply renders this area vulnerable to malignant invasion, exactly as the syphilitic virus can enter a minute crack in a healthy skin.

4. The virus, once established in the affected area, produces biochemical reactions in the cells resulting in the production of the specific chemical factor.

5. That each type of tissue cell, having different chemical constituents, a different specific chemical factor is produced for each type of cell.

6. That this "disordered cell chemistry" is possible only because the normal control of the cell by its trophic nerves is withdrawn.

7. That cancer is painless in its origin because the nerve supply to the invaded area has been abolished before the area has been invaded by the malignant virus.

8. That the absence of pain and glandular infection in basal cancer is due to the protection given to the surface of the ulcer by the persistence of an epithelial covering.

9. That squamous-celled cancer, not having this epithelial protection is invaded with bacteria, becomes septic and invades the neighbouring glands.

10. That the septic inflammation by irritating the nerve fibrils of the inflamed tissue causes the pain noted in this class of cancer.

11. That metastasis does not occur in rodent cancer because the slow aseptic destruction of the edges of the ulcer seals the lymphatics in advance of the ulcerative process.

12. That metastasis does occur in squamous cancer because the septic inflammation produces increased vascularity and offers easy access to the neighbouring glands.

13. That rodent cancer originates in the palisade lining cells of secreting glands, that is the sebaceous

or sweat glands, while the true epithelioma originates in the keratin containing cells of the horny layers of the epithelium.

DR. H. F. PRAAGST (Victoria) showed some interesting films. The series depicted calculus in the right ureter with dilatation of the ureter and hydronephrosis, bilateral polycystic kidney with dilatation of the pelvis, a poor filling defect of the renal pelvis which was filled completely in the second skiagram, multiple polypi of the large bowel with complete involvement, clinically a polypus was projecting from the rectum, a skiagram of the chest showing well developed substernal goitre in a girl suffering from exophthalmic goitre, the thyreoid was subsequently removed surgically. There was another skiagram of a large hydatid cyst of the lung. A skiagram of a rare condition was exhibited. There was a filling defect of the cardiac end of the stomach in a woman who had symptoms of gastric carcinoma; at operation a myoma had been found at this site. A further interesting case was that of a neoplasm of the spine diagnosed with the aid of "Lipiodol" injection; in the antero-posterior view the "Lipiodol" could be seen deflected to the left at the level of the sixth dorsal vertebra, in the body of which there was an area of absorption on the right side with a soft shadow fading into the lung tissue at the same level. The condition was inoperable, but responded well to combined radium and deep therapy treatment. A further film was that of a lung with a small bronchiectatic cavity rendered visible by intralaryngeal injection of "Lipiodol." There was no evidence of tuberculous infection of the lung steroscopically. The patient had swallowed a chop bone sixteen years previously, had suffered from hæmoptysis at short intervals for years and was quite convinced in his own mind that he had pulmonary tuberculosis. From the above examination, however, he was reassured on that point. Moreover, he improved considerably after the diagnostic injection of the "Lipiodol."

A film illustrated an obstruction with dilatation of the duodenum with residue in the stomach; at the operation a mass of tuberculous glands pressing on the third part of the duodenum was found.

DR. W. R. STOWE (Palmerston North) showed a skiagram of a skull in which the semicircular canal was clearly visible.

DR. D. F. MYERS (Wellington) showed a series of films illustrating various pathological conditions of the colon.

WEDNESDAY AFTERNOON, FEBRUARY 9, 1927.

COMBINED MEETING.—SECTIONS I AND VIII.

NEUROLOGICAL ASPECTS OF THE SEQUELÆ OF ENCEPHALITIS LETHARGICA.

By H. F. MAUDSLEY, M.C., M.D. (Melbourne), M.R.C.P. (London), D.P.M. (England),

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SINCE cases of epidemic or lethargic encephalitis were first described in 1918, the attention of neurologists has been drawn to the extrapyramidal motor system. Observers had already begun to realize that the symptoms of *paralysis agitans* were due to degeneration of cells in the *corpus striatum*. The condition of progressive lenticular degeneration had been described in which extrapyramidal symptoms were correlated with striatal degeneration.

Chronic *encephalitis lethargica* is essentially a disease of the extrapyramidal motor system, although owing to the diffuseness of the lesions throughout the central nervous system the neurological symptoms may be most varied. Soon after the earlier epidemics it was said that 50% of the patients who recovered, developed sequelæ. We realize now that this figure is a fallacious one. In cases of apparent recoveries, many months or even years may elapse before the chronic state manifests itself. Wimmer[1] says that: "the encephalitic process may remain active for years as an intermittent and recurrent process which in by far the majority of cases is of a progressive nature."

In the same way that the primary infection in neurosyphilis may take place unnoticed by the patient, so many cases of advanced chronic encephalitis may supervene on an apparently mild attack of influenza and in some a history of primary disease cannot be elicited at all. Wimmer[1] points out the rarity of pulmonary, renal or cardiac after-effects and considers that the organism of epidemic encephalitis has a purely selective influence upon the nervous system.

Zymanowski and Zylberlast-Zand[2] have, however, noted infiltration of liver bile ducts in animal experiments. The exacerbations and remissions that often occur, force us to the opinion that the virus can remain active and inactive in the body over long periods. Reinfection cannot account for the remissions, as Wimmer[1] points out that animal experiments show that an immunity is produced, similar to that produced in anterior poliomyelitis.

The most common extrapyramidal syndrome met with in chronic encephalitis is that of Parkinsonism. This may be very slight in the mild cases or it may be masked by more obvious neurological signs or it may be a predominating feature, clinically indistinguishable from *paralysis agitans*. Bradykinesis, a feature of the Parkinsonian syndrome, is seen in practically every case of chronic encephalitis that I have seen and is the cause of a great many of the psychological symptoms met with in these cases.

Wimmer[1] classifies chronic epidemic encephalitis into: (i.) Cases presenting gross Parkinsonian symptoms (ii.) an intermediate type showing signs of diffuse neurological lesions; these cases practically all show bradykinesis or rigidity in his series, (iii.) hyperkinetic cases with extrapyramidal tremors, forced movements, spasmodic tics and respiratory symptoms.

In 1921 Ramsay Hunt[3] concluded that the striate-spinal system, functioned as a motor pathway for automatic movements. More recently Vercellum[4] in reviewing recent literature came to the same conclusion and considers also that the *corpus striatum* controls plastic muscle tone through the vegetative nervous system, the latter being based upon Hunter and Royle's researches upon plastic muscle tone.

Wilson,[5] on the other hand, produces arguments against the *corpus striatum* having such an important function as that of controller of such complicated movements. He does not think that its anatomical structure and size warrant such an assumption.

Cobb and Wilson[6] describing the condition in patients with mesencephalic syphilis, found lesions in the red nucleus, in whom Parkinsonian symptoms were seen. Lhermitte,[7] however, describing syphilitic Parkinsonian conditions, found a reduction in the cells of the *globus pallidus*.

McAlpine[8] reported fully the *post mortem* findings in cases of postencephalitic cases with Parkinson's syndrome. He demonstrated lesions in the *substantia nigra* in all his cases. In some of them the *globus pallidus* was also slightly affected. His conclusions were that the main incident lesion of chronic encephalitis is in the *substantia nigra*, whereas in *paralysis agitans* the *corpus striatum* is primarily affected with occasional descending degeneration in the *substantia nigra* cells. It is generally agreed that the Parkinsonism in chronic *encephalitis lethargica* is caused by an inflammatory lesion in the *substantia nigra*.

It may be well here to consider briefly some facts concerning the morphology and anatomy of the basal ganglia. The *corpus striatum* consists of a paleostriatal portion, the *globus pallidus* and the neostriatum, the *putamen* and caudate nucleus. The paleostriatum is phylogenetically the earliest correlator for motor activity, the first differentiation from the purely segmental animal. The neostriatum developed later as a controller of paleostriatal activity.

In this early stage of evolution of the motor brain, muscle movement was of the most primitive type, for the preservation of life, in response to the most primitive form of sensory impressions received by the paleothalamus. With the development of the neopallium and the cortico-spinal motor tracts, on the one hand, and the more complex mid-brain centres on the other hand, the *corpus striatum* became of relatively less importance.

The mid-brain has developed very important reflex centres, namely those for postural tone, labyrinthine and cerebellar activities, those for reciprocal innervation as well as those for associated reflex activities exhaustively studied in man by Riddoch and Buzzard.[9]

The continuity of the striato-spinal projection in fibres has been broken by the development of these complex centres. The *corpus striatum* acts now indirectly upon the final common motor path through the *substantia nigra*, the rubro-spinal and tecto-spinal tracts.

One of the clinical features of extrapyramidal dysfunction, as seen in the post-encephalitic sequelæ, is slowness and difficulty in performing voluntary movements and the loss of so-called automatic movements. This is the condition of bradykinesia of which mention has already been made. Walshe[10] has suggested that bradykinesia is directly proportional to and dependent upon the presence of increased muscle tonus. There are, however, cases which present the bradykinetic syndrome without increased muscle tone being present and *vice versa*.

The tecto-spinal tract at its mid-brain end is in close relationship to the posterior longitudinal bundle and probably acts as a coordinator of various muscle groups, in the same way as the posterior longitudinal bundle acts upon the muscles supplied by the cranial nerves. Muskens[11] has pointed out the connexions between the *globus pallidus* and the mid-brain centres for these tracts. This connexion may come through the *substantia nigra*, although anatomical proof of this is wanting. On this hypothesis disease of the *substantia nigra* interferes with the connexion between the *corpus striatum* and the final common path. It is reasonable to suppose that some remnant of the original function of the *corpus striatum* remains. The tecto-spinal tract is a co-ordinating one and we may thus on physiological and phylogenetic grounds presuppose that the *corpus striatum* gives rise through this coordinating tract to a striatal or non-voluntary component of every voluntary muscle group movement. Ordinarily these movements are seen in the so-called automatic movements. Wilson[5] states that striatal disease cannot give rise to a positive loss of function, as there is no true paralysis in Parkinsonism. However, the cortico-spinal tracts being intact, any movement can be performed, although, of course, with far greater difficulty and effort, as there is no non-voluntary component to help the movement. Briefly put, the *corpus striatum* through its connexion with the *substantia nigra*, has a positive action upon the final common path.

The absence of this function is seen clinically in the condition known as bradykinesis. Movements in which the non-voluntary element predominates, are most noticeably affected. Such movements consist of arm swinging when walking, movements of facial expression, vocal expression and blinking of the eyelids.

There is a non-voluntary element in every voluntary movement and loss of this non-voluntary element gives rise to the difficulty in performing voluntary movements in the muscle groups affected. There is no true paralysis, as the cortico-spinal tracts are functioning and can carry out all movements, though with greater effort than normal.

An enthusiastic golfer, who has marked bradykinesis as a sequel to an attack of *encephalitis lethargica*, told me that accuracy of his game has improved since his symptoms had made their appearance. The reason for this can be appreciated when one considers that the ordinary golfer plays bad shots because of the non-voluntary movements in his swing. When this non-voluntary element is eliminated, the movements taking part in the swing are completely under cortico-spinal, that is voluntary control. Provided that he knows the correct method of swinging, his game must become steadier. He will never acquire the automatic swing of the

champion golfer, in whom the voluntary and non-voluntary components of movement are so perfectly blended. This patient had very little rigidity which if present to a considerable extent, would impede such muscle activity.

Bradykinesia produces many of the so-called functional symptoms met with in the sequelæ of encephalitis. This is interesting in view of Golla's[12] theory of the physical basis of hysterical manifestations. He ascribes hysteria as being the result of deficiency in efficient muscular control in response to an emotional stimulus. In chronic encephalitis emotional abnormalities are often found and the explanation of this may be sought in the fact that the non-voluntary element in movement in response to emotion is absent. It must not be forgotten that lesions are sometimes found in the thalamus which may also contribute to these emotional instabilities.

The question of the increased muscle tone met with in Parkinsonism is at present under the consideration of neurologists. We are all familiar with Hunter and Royle's conception of plastic tonus. Their conception embraces the hypertonus under consideration. Hunter[13] considered that the *corpus striatum* has an inhibitory influence upon the reticular nuclei in the pons. These muscles in turn are the mid-brain centres for the efferent sympathetic nervous system. Garven[14] considers that it is doubtful whether non-myelinated nerve fibres are found in every muscle and doubts the existence of the two types of muscle fibres described by Hunter.

Adrian, Bankart and Walshe[15] in a discussion upon the sympathetic innervation of striated muscle, consider that clinical and experimental evidence do not uphold Hunter and Royle's conceptions. Kinnear Wilson[5] points out that the rigidity met with in *paralysis agitans* and allied conditions, cannot be plastic in Hunter and Royle's sense, because of the attitude of flexion taken up by the patient in advanced cases. The centre for postural tonus is in the mid-brain at the level of emergence of the eighth cranial nerve, thought by Hunter to be controlled by the vestibular-spinal tract. Release of striatal control from the nucleus must disturb the normal Deitero-cerebellar-rubral impulses, so that apart from the presence of plastic tone, there may be some disturbance in postural tonus.

That sympathetic innervation to glandular structures is upset is undoubted, when the salivation, greasy skin and other secretory abnormalities met with in this condition are considered.

Walshe[10] has demonstrated by paralysing the sensory nerve ending in muscle substance by injecting cocaine that the tonus is a reflex condition.

The tremors and involuntary movements may be very varied, but it is not common to see the typical pin-rolling tremor of *paralysis agitans* in the sequelæ of *encephalitis lethargica*. A simple tremor of the jaws is, however, common. Walshe in his cocainizing experiment showed that the tremors remained after the rigidity has been abolished; thus these effects are brought about by different mechanisms.

C. and O. Vogt[16] found that in cases of *paralysis agitans* with tremor the *putamen* and caudate nucleus are chiefly involved, whereas when tremor is absent, the *globus pallidus* shows the greater degeneration. This falls in line with the conception that neostriatum has normally an inhibitory influence upon an inherent paleostriato-spinal tremor. Wilson[5] considers that tremors may occur at the lowest and highest physiological levels. The *corpus striatum*, he considers, inhibits an inherent neuromuscular tremor at the spinal level.

The more complicated tremors, forced movements and respiratory spasms seen in the hyperkinetic group of Wimmer's classification, would appear to have their origin in the extrapyramidal system. Wilson postulates an interference with the cerebello-mesencephalo-thalamo-cortical connexions to account for choreo-athetoid movements.

The intermediate type in Wimmer's classification presents the most varied neurological symptoms. There is, of course, constant overlapping in this classification of Wimmer. For instance, McAlpine[17] describes a case in which rigidity, bradykinesia and choreiform movements were present on one side with a pyramidal tract lesion on the opposite side. The pyramidal tract lesion masked the Parkinsonian symptoms on that side, giving an altogether different distribution to muscle tonus and decreasing the athetoid movements.

Cranial nerve palsies are commonly met with, ophthalmoplegia being the most common. Inequality of the pupils is common. I have seen three cases in which the light reflex is preserved with absent accommodation reflex. Wimmer[1] states this to be not uncommon and says that Argyll-Robertson pupil may be present. He mentions, however, that Lhermitte does not consider that the Argyll-Robertson pupil belongs to chronic epidemic encephalitis. In Cobb and Wilson's *mesencephalitis syphilitica* Argyll-Robertson pupils were invariable and in these cases Parkinsonism was found, so that a mistake in diagnosis may occur in cases of this type.

Of other eye symptoms retrobulbar neuritis is occasionally found. There is often a curious condition of partial blindness sometimes encountered with telescopic contraction of the visual fields, a condition much akin to the functional type of blindness. Epileptiform and Jacksonian seizures may be present; the former may be of a so-called functional nature and as in all the functional symptoms of this disease is improved when the environmental difficulties are removed.

Intermittent hemiplegias or monoplegia may suggest the presence of disseminated sclerosis.

The encephalitic process may spread to the bulbar region and even to the spinal cord giving rise to a condition resembling the amyotrophies. I have a patient under observation with considerable wasting and reaction of degeneration of the extensor muscles of the wrist with flexion contraction of that joint.

Respiratory disturbances, such as apnœa, spasmodic coughing, yawning and hiccough, may arise from medullary interference.

The encephalitic process may spread from the *substantia nigra* which is anatomically close to the superior cerebellar peduncles, and may give rise to certain cerebellar symptoms. The cerebellum itself is rarely involved.

Objective sensory changes are rare, but subjective sensations are common. One patient of mine complained of an intensely irritating feeling as though insects were crawling over her scalp. Another stated that if he walked in his bare feet he felt as though he were walking upon sharp points. These subjective changes appear to be of thalamic origin.

There is no need to elaborate further the neurological entities that may be met with in this condition. Time does not allow of discussion on the interesting mental phenomena that take place. There is invariably some change in personality and character. This is most evident in patients under the age of puberty.

In regard to the treatment of chronic epidemic encephalitis we are in the same unsatisfactory position as we are in regard to a number of other chronic neurological conditions. We may temporarily relieve symptoms by the administration of drugs that will depress the mid-brain centres; belladonna, nicotine and hyoscine have all been tried with varying results. Hyoscine hydrobromide (0·6 milligramme) taken by mouth is usually well tolerated over long periods and I have found that this gives the patient most relief. It certainly lessens the excessive salivation that is often seen and it also lessens the rigidity.

I have seen three patients on whom ramisectomy has been done, but with temporary improvement in one only.

I think that one aim should be to prevent the exacerbation and progressiveness of the disease by means of plenty of rest and good hygiene. We have yet to find a specific drug or antiserum that will prevent continuation of the chronic condition before complete destruction of the nervous structures has taken place.

References.

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THE SEQUELÆ OF LETHARGIC ENCEPHALITIS.

By WILLIAM MARSHALL MACDONALD, M.B., C.M. (Edinburgh).
Dunedin.

SOME ten years ago lethargic encephalitis appeared in Europe and was hailed as an interesting discovery. Now, unfortunately, it comes annually and takes a serious toll of our youth and manhood and is playing a considerable part in filling our homes for incurables.

We are dealing today with the sequelæ and are at once faced with the difficulty of defining them, as many so-called sequelæ are in reality evolutional processes of the original disease. In syphilis the primary infection may be so slight as to escape notice and yet may be followed later by serious nervous complications, formerly called parasyphilitic, but now known from the demonstrable presence of the *Treponema pallidum* in the nerve centres to be syphilis in actual evolution. The parallel with lethargic encephalitis is striking as many cases in which the original illness is of a very slight nature, are followed at an interval of months or even years by so-called sequelæ which are probably continued evolutional states. In lethargic encephalitis there is a protean multiplicity of lesions, almost every area in the central nervous system having been demonstrated to be the seat of attack and yet the disease preserves its personality so to speak and there is a definite symptom-complex from the main features of which the departures are only minimal. This forms another analogy with syphilis because exactly the same thing occurs in general paralysis of the insane. This seems to suggest that an infective disease has a definite

identity or personality which is not solely dependent on the anatomical lesions. It was one of the Mayo brothers who said that only one thing was more misleading than statistics and that was a *post mortem* examination. While then a great deal will no doubt be learnt in the next twenty years from the examination of brains of persons dead of lethargic encephalitis, it is just as well to bear in mind that there may be too slavish an adherence to histological misinterpretation of function.

Some cases are of slow, chronic, almost torpid development and it is difficult to draw the line between disease and sequelæ. Others begin as Parkinsonism and never cease to progress along that line. Still others are monosymptomatic at the onset and remain so in the chronic stages. But as a rule sequelæ of any kind may occur after each form of the acute disease of which the chief types are the somnolent, the agitant, the confusional, the algesic, the myoclonic (or both of these combined), the paraplegic, the chronic, epidemic hiccup and what is known as the attenuated influenzal type. Nor, in dealing with sequelæ, should the larval, ambulatory forms be forgotten as they so often lead to an *a posteriori* diagnosis.

The commonest of all sequels is Parkinsonism which was first described in France and in that way got its English name, because in France they have never called *paralysis agitans* by any other name than *le Parkinson*. In Parkinsonism there is an increase of plastic tone due to some interference with the prespinal reflex arc which passes through the sub-pallidal nuclei in the *locus niger*. The rigidity of Parkinsonism has nothing to do with the tremor which sometimes accompanies it, that is, the tremor is not simply an interrupted rigidity because "Novocaine" can abolish the rigidity and leave the tremor unchanged. The rigidity of Parkinsonism is, therefore, in reality due to a true proprioceptive reflex action and so there may be something in the theory of Ivy McKenzie, that it is caused by degeneration of the vestibular nuclei which was found in experimental rabbits. In an ordinary hemiplegia from a lesion of the internal capsule cortical control is lost and the influence of the *corpus striatum* is retained, so that the patient has loss of voluntary, with retention of emotional or automatic movement. In Parkinsonism on the other hand voluntary movement is retained, but automatic or expressional movement is lost, giving rise to the mask-like face. The path followed by the impulses for expressional movement is through the lens and red nucleus to the cells in the motor nuclei; the knee jerks are not usually increased and there is no clonus or toe extension. When the caudate nucleus is affected, there are gross choreiform or clonic movements.

In addition to the mask, there is slowness of movement, diminished blinking, absence of swaying the arm in walking, slowness of speech, loss of power of delicate movement and in writing, the letters become gradually smaller. There is improvement under the word of command, but fatigue soon comes. Writing to dictation is better done than spontaneously, but the patient soon lays down the pen. Another test is the *pruova della sedia*. The patient is seated in a chair and the chair is suddenly tipped back. A normal or a hemiplegic limb is at once shot up, but the Parkinson limb remains flexed against the chair. The *pruova del mulinello* consists in vigorously circumducting one arm when no synergic movement follows in the opposite limb. Although it is common to say that there is no intellectual change in Parkinsonism and that the psycho-motor time is increased, but not the cerebration time, still there is an undoubted mental apathy and a certain mental apraxia. Postencephalitic Parkinsonism is distinguished from ordinary *paralysis agitans* by the early appearance of the mask, the absence of pill-rolling tremor and of the steady progression from one limb to the other. Other excito-motor phenomena of extrapyramidal origin which occur, are spasmodic torticollis, *dysbasia lordotica* and prolonged myoclonus. Although sensory symptoms are so pronounced a feature of the acute illness, in the form of the so-called root pains which are probably due to vascular changes in the optic thalamus, it is very rare to find sensory sequelæ.

The group of sequelæ that comes second in importance consists of the mental or rather moral changes which occur chiefly in children, such as instinctive perversions, fugues, impulses, changes in temper and character, obsessions and even catatonic and pseudo-schizophrenic syndromes. These children may bark like dogs or cluck like hens, burst into uncontrolled laughter or weeping. They may stammer, remain mute or repeat words (palilalia). They may have periodic attacks of hiccup, sighing, swallowing, sniffling, puffing or blowing, apnœa, tachypnœa and so forth. Nocturnal restlessness is a very common feature. A few of these children become idiots, some of them develope into the *apache* type, while most of them present the problem of the "difficult child." They become aggressive, cheeky, quarrelsome, untruthful, dirty or even obscene. They take to stealing and lying and may develope sexual perversions or even become suicidal. I saw one girl who shut herself up in a room and turned on the gas, but did not kill herself. Most of these symptoms are simply due to a releasing of characteristics which are latent in every child, but are controlled. Direct psychotherapy is of little use in these cases, but much can be accomplished by careful parental control and the establishment of an *esprit de cure*. In adults the mental change may take the form of depression or hypochondriasis. They become egocentric to a degree or in the case of women uterocentric and they develope a peculiar pose and a vacant expression. The moral sequelæ are of special interest to those who are occupied in medico-legal and pension work.

Injections of milk sometimes calm the nocturnal restlessness, but just as often excite the patient still further. Belladonna in large doses should always be given a trial and it should be combined with hyoscyamus. In excito-motor cases hyoscine should be used along with injections of magnesium sulphate.

In conclusion I wish to emphasize the striking resemblance between the mental sequelæ of lethargic encephalitis and the symptoms that occur in the ordinary psycho-neuroses. Some of these sequelæ, such as sobbing, crying, uncontrolled laughter, are part of the normal life of the infant. Others, such as perversions, fugues, hysterical states, convulsions, occur independently of encephalitis and they all tend to show a rhythmical periodicity which is common to the psycho-neuroses. In fact, the whole *ensemble* of the clinical picture in its functional discordance, its irregularity of evolution, its strange intermissions and its paradoxical associations assumes the veritable lines and shades of a psycho-neurosis. Especially does it resemble those neuroses which persist in spite of all treatment. The diagnosis of an organic lesion may rest solely on the history of a previous attack of encephalitis and if this is lacking, we fall back on hysteria, neurasthenia, psychasthenia. What in effect are hysteria, neurasthenia, psychasthenia? In the light of this new knowledge and in view of the organic changes in encephalitis resulting in such intangible phenomena as the mentality of the "difficult child," may it not be possible that the psycho-neuroses themselves are produced by excitations and inhibitions due to vascular changes in the mid-brain consequent on naso-pharyngeal infections, fevers or influenza? This is only an hypothesis, not even a working hypothesis, but it may prove a fertile field for histological investigation.

LOCALIZATION OF SPINAL TUMOURS.

By J. F. MacKeddie, M.D. (Melbourne).
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For every spinal tumour, medullary, subdural or extradural, that makes its exact location known by neurological signs, a great number at any rate in their early history conceal their position behind a cloud of doubtful neurological findings. Too often the surgeon has been invited to cut down on a tumour that was found, if at all, only by largely extending the laminectomy up or down or in both directions. Spinal surgery has often been undertaken in too exploratory a fashion. Moreover, apart altogether from localization, the presence even of spinal compression has gone undetected in some cases for years.

Any method of limiting the surgical procedure in spinal cases to an exact field must be of great value and it is significant that it was the leading neurologists at home and abroad, men who could be expected to locate a compression spot on the cord exactly, who welcomed and put into quick practice Sicard's method of spinal localization.

As is well known, Professor Sicard chanced on a substance that was opaque to X ray, harmless to tissue and of a greater specific gravity than cerebro-spinal fluid. This he injected into the *cisterna magna* and found that it slowly sank down the theca and in normal subjects found lodgement in the *cul-de-sac* at the second sacral vertebra; any compression held it up, in part or whole.

Prior to Sicard's work Froin had demonstrated that cerebrospinal fluid below a compression point gave certain characteristics that were almost pathognomonic of that condition. The presence of Froin's syndrome therefore came to be regarded as an almost certain indication of compression, so that just as formerly a lumbar puncture might have decided the question of operation, it now decides the question of intrathecal injection of "Lipiodol." It can be only very rarely that "Lipiodol" will be held up for any length of time in the absence of a Froin's syndrome. This will be referred to again in a case in which a temporary hold-up in the absence of Froin's syndrome was made an excuse for operation.

The harmlessness of "Lipiodol" has been amply confirmed by all who have used it. Its usefulness in spinal work is now beyond all question and one has only to add the surgeon's comfort in doing a laminectomy with the X ray film in front of him, his objective clearly defined, to illustrate a further and and not the least helpfulness of this diagnostic agent.

I shall now consider the steps taken in *cisterna magna* puncture.

The first landmark to be demonstrated is the spinous process of the axis. Assure yourself of its position before laying the patient down. You may put a pencil mark on the skin corresponding to the point of this process, if there has been any difficulty in placing it. Usually you will be able to demonstrate it quite easily. Another point that it is advisable to locate while the patient is sitting up, is the middle point of the neck. The lying posture may distort this and if it is a fat neck, you may not be quite sure of being in the middle line. Put a small mark just under the external occipital protuberance.

The patient is laid on the left side with the head slightly flexed. You must see that the head is placed higher than the neck and shoulders. This is of no importance in so far as the cisternal puncture is concerned, but if "Lipiodol" is to be introduced, it must get its initial start downwards. It has occurred that the "Lipiodol" has found its way into the subarachnoid space at the base of the brain and did not begin its descent for some days without, however, doing any harm.

If the finger is placed on the spinous process, the point of puncture is immediately above and close to the finger. This spot will be exactly in the mesial plane. Of this you have made sure before laying the patient down. If you are in doubt at all of the little depression just above the spinous process of the axis, you may confirm it in this way. Lay a flexible metal tape measure on the glabella and the upper part of the external auditory meatus. Lay it down so that it extends backwards nearly to the middle line of the neck; it will point directly into

this supra-axis depression. You may, if you like, mark on the neck the line of the tape measure that reaches backwards from the ear. This gives you the exact plane in which the needle will travel. All this is done with the head at slight flexure. The marks that you have made, are not included in the actual field of the puncture, so that you can sterilize without erazing them.

You may anæsthetize with "Novocaine," first raising the intradermal bleb and then passing through this bit by bit in the direction in which your puncture needle is to go, but never penetrating deeper than three centimetres. Sir James Purves-Stewart always freezes the skin and goes right through without any further anæsthetizing and without any pain to the patient. With the former method you can be always sure of giving no pain whatever.

You have made sure of your entry point and pushed the needle, say, one and a half centimetres into the neck. Free the needle well so as to have complete control of it. The further course of the needle is the junction of two planes, the mesial plane and a plane running through the entry point, the upper part of the external auditory meatus and the glabella. When you set the needle in those planes, it will not be perpendicular to the surface of the neck; the point will look slightly upwards. You push your needle straight through the middle of the neck at the same time directing its course in the glabello-meatal plane. A lead pencil placed erect in the ear will emphasize the glabello-meatal plane. At first, seeing that you have your attention fixed on the glabello-meatal line, there is a tendency to point the needle at that line; on stopping near your objective to take stock, you are surprised to find your needle out of the mesial plane. However, this is easily overcome by practice.

You can push fearlessly on up to three centimetres, provided you are in the right line. You are not going to tap the *cisterna magna* under 3.5 centimetres. At this point withdraw the stilette and push on carefully. In all probability you will feel the resisting occipito-atlantal ligament which you will distinguish quite easily from bone.

A thrust will tap the fluid. If no fluid comes, use the stilette to clear the needle. If still no fluid, press on with a screwing motion, using the stilette frequently. You may be at the 4·0, 4·5 or 5·0 centimetre mark. Make sure at this stage of your direction being absolutely true. You may reach the 6·0 centimetre mark or more before success. In the great majority of cases round about 4·5 centimetres the fluid appears. Meantime, see that the patient has not moved his head from the semi-flexed position.

All this detail, of course, is for the beginner. Sir James Purves-Stewart lays his left forefinger on the spine of the axis, turns the ethyl chloride spray on to the skin immediately above his finger point, puts a lead pencil erect in the ear of the patient and with his eye on the pencil and the glabella, passes his needle straight into the *cisterna magna*.

Preparation of the Patient.

The bowels should have been well opened. If the puncture is to be made in the forenoon, only something light to eat should be given first thing in the morning.

No morphine is required. It is a perfectly painless procedure and the withholding of food till late in the afternoon frees it from any untoward symptoms. The patient is shaved from the external protuberance downwards.

Needles.

We find it convenient at the Alfred Hospital to have very conspicuous marks on our needle at the maximum limit. Thus we have three needles, one has a small ring of solder round it at five centimetres, in another the solder is at six centimetres and a third has its ring limit at seven centimetres. All the needles are marked off in centimetres from the point to the solder ring. This prominent mark serves two useful purposes. It keeps your limit strikingly before you and is an easy mark from which to glance along and see how many centimetres you have yet to spare. The thickness of the neck, of course, determines the choice of needle.

An ordinary lumbar puncture needle of medium size does very well. If it is too thin, it will not carry the graduated marks well. Besides "Lipiodol," even when warmed, is viscid and will not readily pass along a needle of small bore.

Meantime your ampoule of "Lipiodol" has been lying in hot water. You break the ampoule into a small receiver and draw liquid into the barrel of a small hypodermic syringe. When a couple of cubic centimetres have been collected from the cisternal fluid, the "Lipiodol" is very slowly propelled into the cerebro-spinal fluid, followed by a little saline solution to clear the "Lipiodol" out of the needle.

Should the cerebro-spinal fluid cease to flow after a cubic centimetre or two has run out, do not put the "Lipiodol" in. Sometimes the needle slips out of the *dura mater*. Draw a little fluid into a small syringe to make sure before delivering the "Lipiodol."

Leave the patient moderately propped up. Make his head comfortable and do not disturb him for an X ray picture for two hours at least. A second picture is taken on the following day.

With some slight variants the above is the routine method of cisternal approach. Sicard, for example, used to have a small syringe with saline solution attached to his needle and as his objective was approached, he tried to sense his entry into the *cisterna* by the ease with which he could discharge the saline solution. We have tried this and found that the saline solution could be injected into the loose tissue about the *foramen magnum* without any appreciable obstruction, with the result in one instance that a patient is now carrying about some "Lipiodol" in that neighbourhood—without any harmful effect.

(To be continued.)

ILLUSTRATIONS TO THE ARTICLE BY DR. A. J. HALL.

FIGURE I.
Ætatis ten years.—Scleral wound by glass six days before. Marked purulent iridocyclitis and retinitis. There is slight infiltration of the chorioid by round cells in the anterior part. The other exudate is of polymorphonuclear type. No granulation tissue formation.

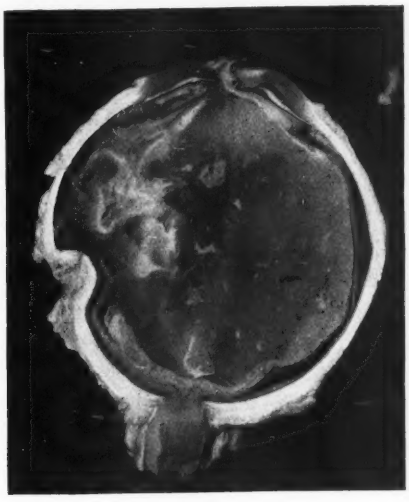

FIGURE II.
Ætatis nineteen years.—Wound of cornea and lens three days before. The main bulk of the lens is absent and capsule is drawn up into the wound. Layer of white exudate covers the iris, ciliary body and retina. The exudate is thickest in the circumpapillary area of the retina. The chorioid is congested but not infiltrated, polymorphonuclear-fibrinous exudate, worst in ciliary body and retina, iris least.

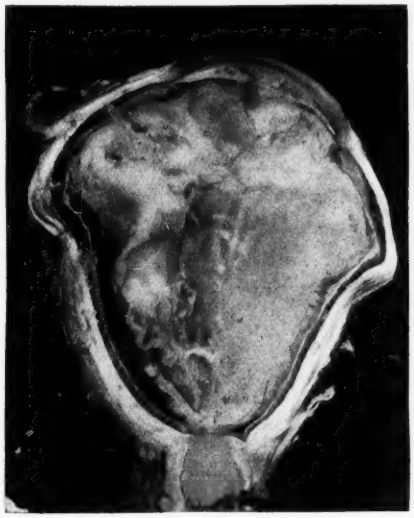

FIGURE III.
Ætatis forty-five years.—Acute panophthalmitis. Large wound of cornea twenty-eight hours before. Purulent infiltration of the iris ciliary body and retina and chorioid.

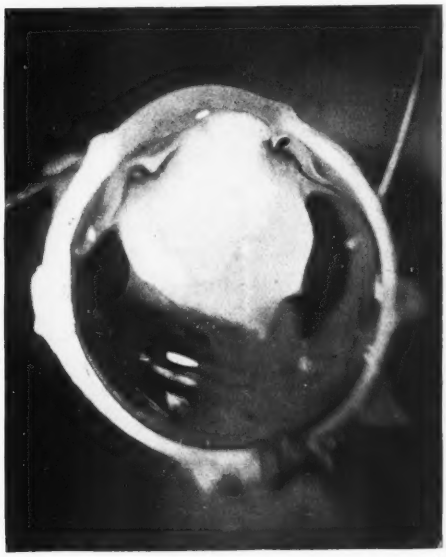

FIGURE IV.
Ætatis ten years.—Wound by knife eleven days before; cornea and lens wounded. Large mass of pus occupies the region of the lens and the anterior vitreous. It has been formed by the shrinkage of the infiltrated vitreous. Newly formed tissue on the ciliary body shutting off the abscess. The retina is infiltrated with round cells and shows early separation changes (folding) from contraction of new tissue on inner surface.

ILLUSTRATIONS TO THE ARTICLE BY DR. A. J. HALL.

FIGURE V.
Ætatis seven years.—Punctured wound of the cornea one month previously. There has been a wound of the lens and the infection has spread from there to the vitreous. There has been a purulent reaction of the ciliary body and the vitreous has been totally infiltrated. The condition is now subsiding and cicatrization taking place. There is a membrane from the posterior part of the ciliary body behind the lens and the vitreous and the retina are shrunken. In the half photograph the part of the retina next to the disc is ruptured; this occurred in preparation.

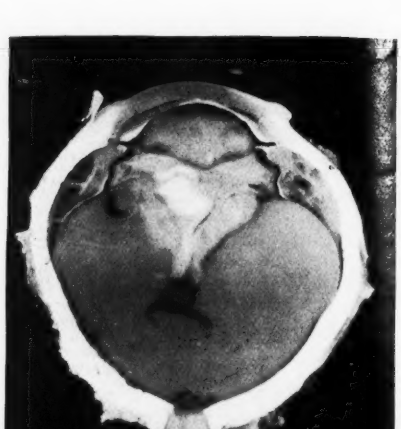

FIGURE VI.
Ætatis eighteen years.—Wound of the cornea eleven months previously. The condition is the late result of vitreous abscess. At the centre of the separated retina are the remains of the abscess. It is encapsulated in a layer of fibrous tissue formed on ciliary body and on the surface of the retina. Subretinal space is filled with gelatinized exudate. Retina torn from nerve.

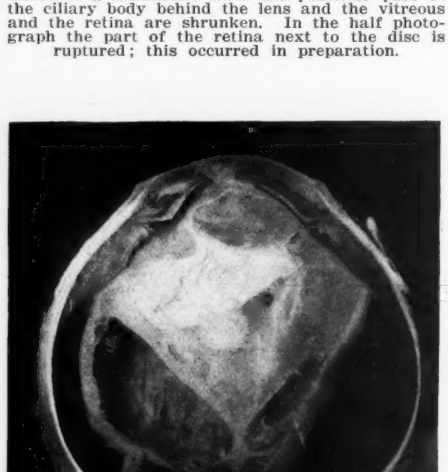

FIGURE VII.
Ætatis fifty-six years.—Wound of the cornea by coal forty-three days previously. Corneal wound to which iris and lens are adherent. There has been a purulent irido-cyclitis and the vitreous is infiltrated and shrunken. The hyaloid has separated from the retina; the area of firm attachment at the head of the optic nerve is seen; the retina is thick and separated; the ciliary body is detached.

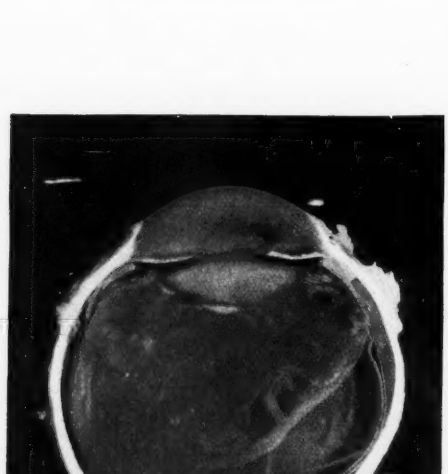

FIGURE VIII.
Ætatis fourteen years.—Penetrating wound of the cornea by wire eleven days before. The wire passed through the iris avoiding the lens and wounded the retina. Thin layer of hypopyon of the cornea and shreds of fibrinous exudate are seen. Cloud in the vitreous most marked over the ciliary body, but also seen in the separated parts adjacent to the retina; the nerve is swollen. Microscopically these are confirmed and it is seen that the inner retinal layers are infiltrated with round cells and the vessels are sheathed in cells.

ILLUSTRATIONS TO THE ARTICLE BY DR. A. J. HALL.

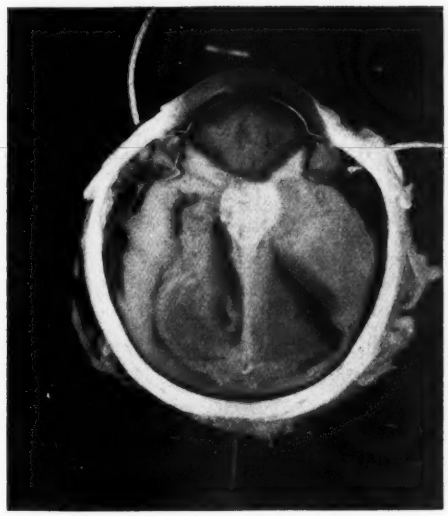

FIGURE IX.

Ætatis ten years.—Penetrating wound of the cornea six weeks before by wire. Irido-cyclitis and hypopyon; the anterior chamber became shallow. The iris adheres to the lens which is pressed forward against the cornea by the fibrous cyclitic membrane. The ciliary body and retina are detached. The vitreous is shrunken and infiltrated, giving the appearance of a small vitreous abscess. Gelatinized subretinal exudate.

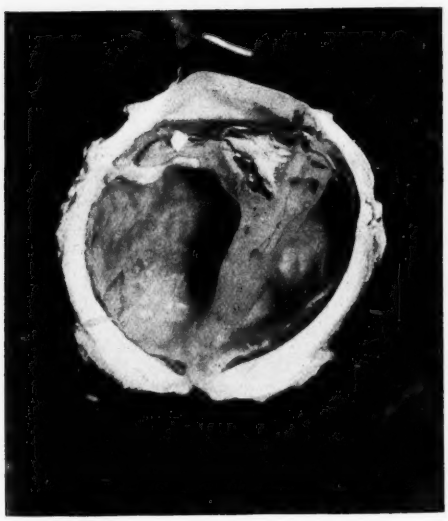

FIGURE X.

Injury to eye thirty years before. A foreign body was seen in the skiagram. The foreign body is seen lying in the mass of fibrous tissue which represents the lens and cyclitic membrane, adherent to in the separated retina. The separation is in the last stage; it is pulled to one sdie presumably by the irregularity of the reaction of the ciliary body.

FIGURE XI.

Ætatis twenty-five years.—Wound of the cornea eight years before by steel. The lens has been injured and the retina is completely separated and adheres to the remains of the lens. In this mass of fibrous tissue there is an area of rust staining which indicates the site of the metal. A gelatinized subretinal exudate has been removed from one half. Microscopical examination shows that the atrophic iris adheres to the cornea and that the space between it and the cyclitic membrane is lined with pigment cells. No sign of lens. Retina adherent to the cyclitic membrane and greatly atrophied. Colloid bodies on chorioid.

ILLUSTRATIONS TO THE ARTICLE BY DR. A. J. HALL.

FIGURE XII.
Ætatis eight years.—Wound across the cornea with sardine tin opener thirty days before. The lens has been wounded and the wounded edges and the vitreous caught up into the corneal scar. Ciliary body wounded near the *ora serrata*. Irido-cyclitis of the plastic type followed and the mesoblastic tissue in the wounded ciliary body proliferated, thus forming the two bands of white fibrous tissue passing up to the wound. The periphery of lens capsule remains in approximate position. Peripheral anterior synechia grooving of cornea along the scar. The iris has thin membrane on surface. The shrunken vitreous is blacker than it should be; perivascular infiltration in the retina; thin layer of cells on the surface and the retina is pulled on to the ciliary body.

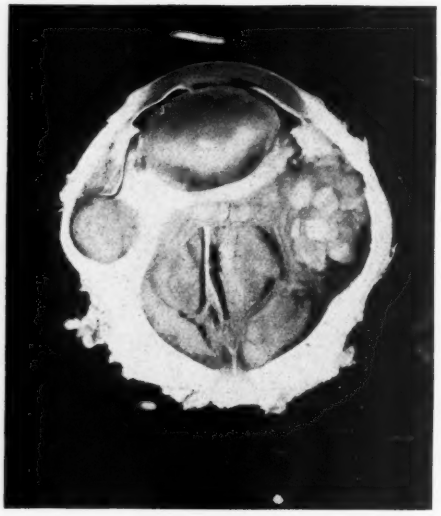

FIGURE XIII.
Ætatis forty years.—Wound of the eye six months before. There has been a scleral wound from which fibrous tissue has proliferated to join the dense cyclitic membrane extending behind the lens. The retina is separated and drawn up to the back of the lens. The ciliary body is detached. The iris is not adherent to the lens, but the lattter has been pushed forward against the cornea. The globe is shrunken. Microscopically it is confirmed. There is in addition a membrane on the inner surface of the retina and the perivascular tissues are increased in thickness.

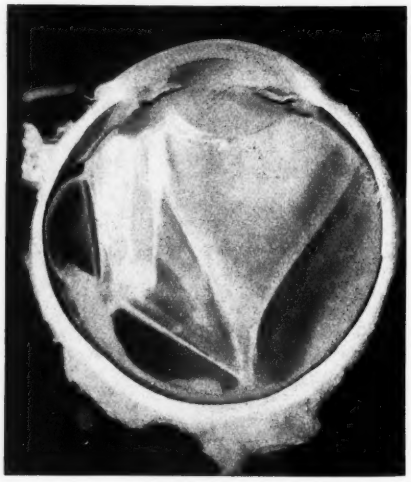

FIGURE XIV.
Ætatis twenty-two years.—Penetration of the limbus by steel chip twenty-two days before. There is seen to be a cyclitis. There is a layer of new formed tissue on the nasal side of ciliary body. The vitreous is cloudy and separated. From the region of the wound a track of young fibrous tissue passes to the point of the retinal wound. It must have been by this route that the metal entered and was extracted. Folds of retina at macula are well seen. The iris shows little change, but the ciliary body is disturbed, largely in the *pars plana*. Round-celled infiltration of retina in the inner layers and early folding.

ILLUSTRATION TO THE ARTICLE BY DR. W. MOPPETT.

FIGURE III.
Effect at 0·79 Angström Units.
Atrophy least intense.

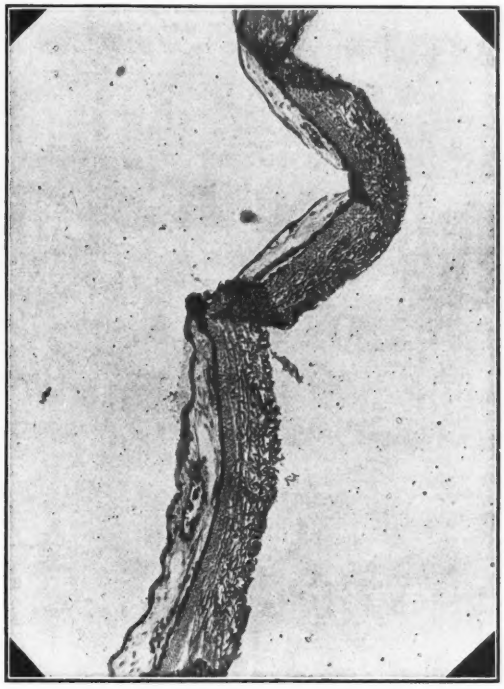

FIGURE IV.
Normal (× 125).

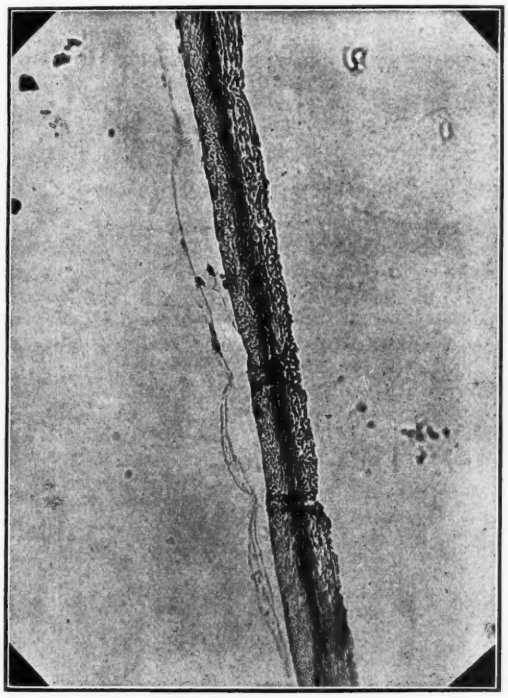

FIGURE V.
Effect at 0·53 Angström Units.
Complete Atrophy (× 125).

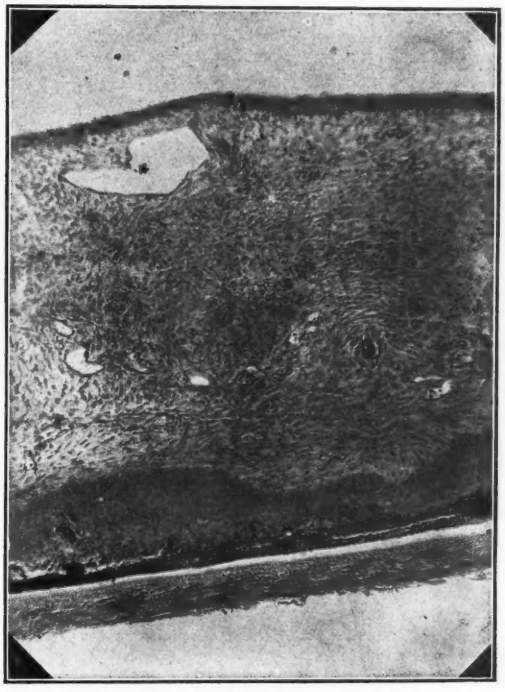

FIGURE VI.
Effect at 0·89 Angström Units.
Peak of Epithelial Stimulation, Shell Membrane below.

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